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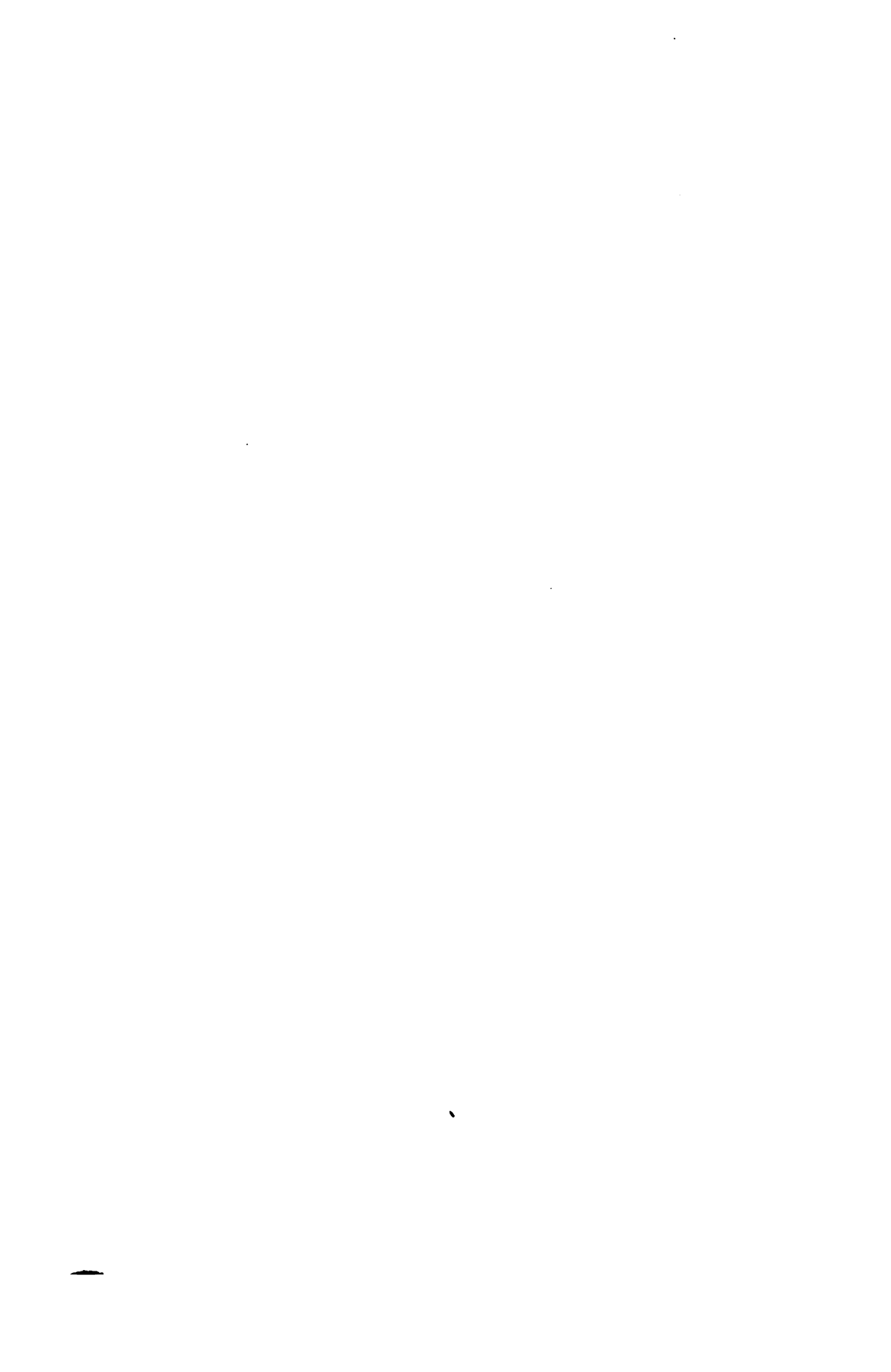


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The Journal

OF

Nervous and Mental Disease

AN AMERICAN JOURNAL OF NEUROLOGY AND PSYCHIATRY
FOUNDED IN 1874

OFFICIAL ORGAN OF
The American Neurological Association
The New York Neurological Society
Boston Society of Psychiatry and Neurology
The Philadelphia Neurological Society and
The Chicago Neurological Society

MANAGING EDITOR AND PUBLISHER
DR. SMITH ELY JELLIFFE

41 North Queen Street, Lancaster, Pa.

64 W. 56th Street, New York City

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Volume 42, 1915

NEW YORK
64 West 56th Street
1915

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PRESS OF
THE NEW ERA PRINTING COMPANY
LANCASTER, PA.

ORIGINAL CONTRIBUTORS.

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The Journal OF Nervous and Mental Disease

An American Monthly Journal of Neurology and Psychiatry, Founded in 1874

Original Articles

SPINAL DECOMPRESSION IN MENINGOMYELITIS¹

BY ALFRED S. TAYLOR, M.D.

CONSULTING SURGEON TO HOSPITAL FOR RUPTURED AND CRIPPLED N. Y.,
PROFESSOR OF OPERATIVE SURGERY, CORNELL UNIVERSITY

AND J. W. STEPHENSON, M.D.

CHIEF OF CLINIC AT THE NEUROLOGICAL INSTITUTE, CLINICAL ASSISTANT IN
NEUROLOGY AT VANDERBILT CLINIC OF NEW YORK CITY

This paper is presented not so much to urge radical surgical intervention in all cases of meningomyelitis, as it is to record such facts and observations as we have been able to collect in a very small series of four cases in which operation was done, and in three of which the results were strikingly satisfactory, in a disease which has heretofore been considered the strongest contraindication to surgical attack. These results raise the question whether in selected cases, decompression will not only greatly shorten the period of invalidism but also very considerably enhance the completeness of recovery.

That this disease is considered to contraindicate surgical intervention is strikingly illustrated by the fact that, in a careful search of the literature since 1907, Dr. Stephenson was able to find record of only one case in which operation had been done. This was done in 1909 by Krause and the clinical history, the operative

¹ Read at the fortieth annual meeting of the American Neurological Association, May 7, 8 and 9, 1914.

findings, and the principles of operative treatment were nearly identical with those in our small series, although we did not know of his case until nearly a year after our last case had been operated upon.¹

Except in syphilitic cases the usual outlook in non-fatal cases of meningomyelitis is for a tedious illness followed by slow cessation of active symptoms and the persistence of a greater or less degree of invalidism according to the site and amount of permanent damage to the cord substance.

It will conduce to brevity to record the case histories and then to make such remarks and draw such inferences as may seem to be warranted.

At the outset we wish to make acknowledgment to Dr. A. R. Diefendorf, of New Haven, for giving one of us (Taylor) the privilege of operating upon the first two cases, and for kindly permitting us to incorporate the two cases with his notes in this paper.

CASE I.—Thos. K. 40 years. Teamster. (Referred by Dr. Diefendorf.)

Previous History.—No evidence of tuberculous or specific infection. He is married and has had eight children of whom five are still living and healthy. At 18 years he had gonorrhea.

Present Illness.—In February, 1910, he suffered from paroxysmal stinging pains, just above the right Poupert's ligament, which appeared on rising mornings and lasted for about ten minutes. There was also pain at the outer side of the left knee when he started to walk.

In November, 1910, instead of the above pains which had disappeared for a short time, he developed below the ribs on both sides severe lancinating pains which shot both forward and upward. These were attributed to pleurisy and disappeared under treatment.

In February, 1911, after exposure to bad weather, he went to bed with a recurrence of the pains accompanied by a cough with expectoration. From February 7, the pain became "band like." On February 14, 1911, while in bed he noticed that his legs were weak. On February 18, the legs ceased moving, and abdomen became like a drum. On February 20, catheterization became necessary. On February 23, incontinence of feces developed. February 24, 1911. Physical examination (Dr. Diefendorf).

Hypesthesia to pain, temperature and touch from the toes to the level of the twelfth dorsal segment. There are no distinct areas

¹ Krause, "Surgery of the Brain and Spinal Cord."

of analgesia. In some areas, especially on the right side the temperature and pain changes coincide in level, while the tactile changes are always one to two inches lower than the pain which is uniformly the highest.

Paralysis is complete from the knees down. The thigh muscles have slight power, the adductors being the strongest. The abdominal muscles are powerless.

Absent on both sides Achilles reflexes, Babinski's sign, testicular and umbilical reflexes.

Present on both sides normal knee jerks.

Firm pressure over the tenth dorsal spinous process causes stinging pain elicited over no other spine.

March 1, 1911. The patient is tired, irritable, and suffers from loss of memory. There is now complete loss of the right knee jerk, partial loss of left knee jerk, and complete paralysis of the thigh muscles including the adductors. The upper level of sensory disturbance is now at the tenth instead of the twelfth dorsal segment. The tenderness over the tenth dorsal spine is much increased.

For the last two days the evening temperature has reached 102° F., but has been normal all day.

There is a small bed sore over the coccyx.

March 3, 1911. Operation under ether by Dr. A. S. Taylor. Right unilateral laminectomy involving D ix, x, and xi was done. When the dura was opened there was no excessive tension of the spinal fluid present, nor was there any feeling of tumor. The whole dorsum of the cord was easily seen. The cord was intensely congested, and seemed somewhat swollen, but showed no irregularity in outline. The right anterior surface of the cord was easily brought into view by means of grasping the dentate ligament and so rotating the cord. The anterior surface showed the same conditions as previously described. The dura was sutured with a continuous catgut suture. The remainder of the wound was closed without drainage by layer sutures.

Following operation there was no change for the better in the neurological symptoms, the cystitis became aggravated, the general condition failed, and the patient died, March 27, 1911, 24 days after operation.

CASE 2.—Mike S. 48 years. Steel Roller. (Dr. Diefendorf.)

Previous History.—Married 20 years. Has four children living, 19, 18, 17, and 9 years old. No children dead and no miscarriages in the family. At 27 years of age had a small sore on the penis which healed promptly under local treatment. For years has taken beer and a couple of whiskeys every day.

Present Illness.—Three months ago (November, 1912), he developed pain over the lower ribs in the axillary line of the right side. He continued at work until about January 20, 1913, when he remained one day in bed because of great weakness in the legs.

Return to work was followed by the development of increasing pains in the mid lumbar region and below the knees as well as by increasing weakness. About January 27, 1913, he took to his bed for two days and developed "girdle sensation." There was no loss of bladder or bowel control.

Physical Examination, February 10, 1913.—Hypesthesia to pain and touch on right side to a level 1 inch below the umbilicus; less marked on the left side and to a level 2 inches below the umbilicus. No zone of hyperesthesia could be detected. Both knee jerks were increased, there was a Babinski on both sides, right ankle clonus was present. The cremasteric and umbilical reflexes were diminished. There was partial loss of power in both lower extremities, more marked in the right. There was marked tenderness over the twelfth dorsal spine.

February 19. There was obstipation and retention of urine. Paralysis of the lower extremities had become complete. Complete anesthesia for touch and pain to a level 3 inches above the umbilicus. The reflex of defense was present on both sides; the knee jerks were exaggerated; double Babinski was present as well as right ankle clonus. There was slight tenderness over the fifth to ninth dorsal spines.

February 19, 1913. Ether. Operation by Dr. A. S. Taylor. Right unilateral laminectomy was done involving D iv, v, and vi. The dura was divided longitudinally the length of the wound and showed nothing specially pathological. The cord was very greatly congested and apparently swollen, somewhat more on the right half of the cord. A longitudinal incision about 1 cm. long and .3 mm. deep was made in the posterior column a little to the right of the median fissure. This developed no evidence of an intramedullary tumor or cyst. There was but very slight bleeding as the result of this incision, and it soon ceased entirely. The dura was loosely closed with the fine catgut sutures and the muscles by layer sutures without drainage. Healing was by primary union.

March 2, 1913. He has regained sphincteric control. Tactile and pain senses, save for a slight qualitative change, have returned in the lower extremities up to a level 1 inch below the umbilicus on the right and 2 inches below on the left side.

April 18, 1913. Very slight sensory reduction to the level of the sixth rib anteriorly. Motion: Right thigh shows slight power of extension, abduction and adduction. There is very slight power to extend the toes. The left lower extremity has regained all movements. Right knee jerk is slightly exaggerated, right Babinski is present but no ankle clonus. Left knee jerk greatly exaggerated, left Babinski is marked, but there is no ankle clonus. All the muscles respond to faradism. This patient moved shortly after this last examination and was lost to observation. He was said to have died in the summer of 1913, but nothing was known as to the cause of death.

CASE 3.—Jacob G. 62 years. Watchman.

His previous history was negative except for an attack of pleurisy and pneumonia in 1905. There was no history of lues or of a cough. His work subjected him to great exposure to cold.

Present Illness.—While at work in May, 1912, he was seized with intense pain in the back and chest. The back became very tender to touch. These pains persisted for several days, he was delirious and was said to have had fever. A few days after the onset an abscess developed over the second chondro-sternal junction on the right side. This was evacuated and the patient's pains were somewhat relieved. At the same time there developed retention of urine and swelling of the scrotum, but these conditions were of only temporary duration.



FIG. 1. Jacob G. Before operation. Note the infiltration on both sides of the lower cervical column obscuring the outlines of the transverse processes. Note the narrowing of the cervical canal from below upward. (These showed more distinctly in the plate than in the print.)

Pain and fever recurred at irregular intervals.

Early in August, 1912, the pain seemed to locate in the back of the head and the neck and this was soon followed by heaviness of the legs and difficulty in walking. The legs gradually became stiff and in place of the pains he began to have cramps which would double him up. Later he also noticed that the upper extremities were growing weak and stiff. About the middle of August he became bedridden. He had retention of urine with

dribbling overflow, and obstinate constipation. On November 13, 1912, he entered the Neurological Institute, Second Division, after having been bedridden for three months, and suffering from severe paroxysmal cramps in the legs.

Physical Examination.—The patient was a short thick necked man, fairly well nourished. There was complete spastic paraplegia. There were no distinct atrophies though the leg muscles were flabby. The knee jerks were plus and equal; the ankle jerks were plus and equal; there was double Babinski and Oppenheim. There was double ankle clonus, the right inexhaustible, the left exhaustible. The epigastric and abdominal reflexes were very sluggish.



FIG. 2. Jacob G. About 16 months after operation. The infiltration on the lateral aspects of the lower cervical spine has almost entirely disappeared. The loss of bone due to the hemilaminectomy is seen just to the right of the spinous processes. It shows most clearly in this print opposite the C VII and D I.

The muscles of the upper extremities were so weak that he could not feed himself. The reflexes of the upper extremities were plus, the left being greater than the right.

There was diminution in thermal, pain, and tactile sensibility up to the level of the second dorsal segment.

There was no tenderness over the spines of the vertebræ but at the base of the neck alongside of the three lower cervical ver-

tebræ there was an infiltration of the soft tissues which was tender on pressure and limited the free mobility of the neck.

Over the second right chondro-sternal junction, at the site of the oldest abscess, there was an infiltrated area about 3 cm. in diameter which was very slightly tender.

The bladder was distended and the colon impacted.

Examination of the eyes was negative except for slight irregularity of the pupils.

Temperature on admission 103° F., P. 68, R. 18; blood pressure 140 mm.; R. B. C. 4,788,000; leucocytes 9600; neutrophils 62 per cent.; large lymphocytes 15 per cent.; small lymphocytes 22 per cent.; transitionals 1 per cent.

The Wassermann test, the von Pirquet test, and the urinalysis gave negative results.

The spinal serology was negative except for a very marked increase in the globulin content.

The patient was kept under observation for 28 days in the hospital, during which time the temperature subsided to normal, but the other symptoms gradually progressed. Areas which showed only a sensory diminution previously, became anesthetic; the abdominal and epigastric reflexes disappeared, and the left exhaustible ankle clonus became inexhaustible. The cramp-like pains became more severe and frequent requiring the use of morphine.

Frequent catheterization became necessary and the bowels were evacuated only by enemata.

An X-ray picture showed a fusiform shadow involving the transverse processes of the C v, vi, and vii on both sides of the spine. This fusiform shadow corresponded in situation with the tender infiltration at the base of the neck previously mentioned. It also showed a symmetrical narrowing of the cervical canal from below upward to the level of C V.

December 10, 1912.—Ether. Operation by A. S. Taylor.

A right unilateral laminectomy was done from C V to D ii inclusive. The bone structure showed no macroscopical pathological condition, but the dura underneath was considerably thickened and congested. It filled the canal tightly. There was obvious narrowing of the bony canal as indicated in the X-ray picture. The inner surface of the bones was smooth and the condition was evidently of congenital origin. The dura was split the length of the wound. The cord, thus exposed, did not pulsate, was much congested and somewhat edematous, and so filled the dural canal as almost entirely to prevent the flow of spinal fluid.

The posterior column, just to the right of the median line, was incised longitudinally to a depth of 2 mm. for a distance of 3 cm. After a brief interval of observation the cord began to pulsate.

The dura was left unsutured. The remainder of the wound was closed by layer sutures. No drainage was used.

The wound healed by primary union.

Within four days after operation ankle clonus had again become exhaustible; and pin pricks were acutely felt over areas which were previously anesthetic. Within eight days he was able to move his legs voluntarily. Within sixteen days he walked the length of the ward with some assistance. The sphincteric control had become nearly perfect. He had an occasional cramp in his legs but never required an opiate.

Six months later he was walking daily to the park, a distance of half a mile, and in addition had three flights of tenement house stairs. This rapid recovery occurred after he had been confined to his bed for four months.

April 2, 1914, not quite sixteen months after operation, an examination gave the following results:

Station is good. Walks quickly and well. There is possibly the slightest suggestion of spasticity.

Reflexes: Abdominals and epigastrics are present but exhaustible. Cremasterics are not elicitable. The knee jerks are present, equal, and not exaggerated. The ankle jerks are present and equal. No clonus on either side. The plantar responses are flexor. No Oppenheim or Gordon reflexes present. In the upper extremity the responses are equal on the two sides, but are slightly exaggerated. Pupils react well to light and accommodation. There is slight atrophy of the thenar and hypothenar muscles on the right side.

Sensation is present everywhere to all forms of stimuli, but there is hyperesthesia below the level of the first dorsal segment.

The sphincters are under complete control.

The tender infiltration at the base of the neck which was present before operation has entirely disappeared, as has the induration at the site of the abscess beneath the clavicle. The neck moves with perfect freedom. Subjectively he complains of occasional pains about the shoulders, numbness of the fingers especially of the right hand, and numbness and coldness of the lower extremities, especially below the knee. (Compare with sensory tests.)

He is in condition to return to work.

CASE 4.—Jacob O. 41 years. Laborer.

His previous history was negative. He has never had any venereal disease. He is married and has six children living and well.

Present Illness.—About March, 1910, he was struck on the right hip by a car fender. In a short time he returned to his work without any loss of function in the hip. From the time of this injury until August, 1912 ($2\frac{1}{2}$ years), he had pains in his right hip and thigh which he thought to be rheumatic. These pains appeared at irregular intervals and were often quite severe, but up to August never extended below the knee.

In August, 1912, while at work, he felt a sudden sharp pain in the back of the right thigh. This remained for three days and then suddenly disappeared. On returning to work a week later the pain recurred, and after three days disappeared, leaving the back of the thigh numb and insensitive to stimuli. Meanwhile a pain of similar character developed in the right heel. This pain was aggravated by placing the foot on the ground. Near the end of December, 1912, a nail stuck in his right heel and remained there for three days before his attention was called to it by the development of infection and swelling. He was taken to the hospital where the nail was removed and the infection cured. After the infection was gone there remained superficial ulceration and thickened fissured horny skin over the external aspect of the right heel and foot. The pains became rather more severe and he was unable to work during the three months preceding the operation in spite of medication and the use of the various methods of physical therapeutics.

There has been no disturbance of the bladder or rectum, except that micturition and defecation often were painful.

Physical Examination.—He is a well nourished, muscularly developed man. He uses crutches to keep the right foot off the ground.

The pupils are equal, and are normal in reaction to light and accommodation.

Tongue clear, moist and non tremulous. Throat normal.

Examination of the chest, abdomen and arteries shows nothing abnormal.

There is an area of anesthesia about three inches wide starting above at the right tuber ischii, passing downward to about three inches above the knee, where it passes to and down the outer side of the leg, ankle and foot, passing over somewhat onto the outer border of the plantar surface.

There is no paralysis of the muscles but attempts to bear weight upon the right foot cause obvious pain. The muscles on the right side seem softer (disuse) than on the left, but there seems to be no real atrophy.

The reflexes are slightly diminished on the right side.

On the outer side of the foot, extending from the back of the heel forward to the base of the fifth metatarsal, from the outer side of the plantar surface nearly up to the outer malleolus, is an area of skin which is thickened, horny, fissured, scaling and showing granulation areas at the bottom of the fissures. A little back of the center of this area is a small ulcer which has remained since the injury by the nail some three months ago.

March 25, 1913. Ether. Operation by A. S. Taylor.

A right sided unilateral laminectomy was done involving D x, xi, xii and L i and ii. The laminæ and the dura presented no patho-

logical appearance, nor did there seem to be any excessive pressure of spinal fluid on opening the dura.

The cord itself over the entire exposed area was considerably congested and the veins in particular were unusually distended.

There was no evidence of tumor or hemorrhage, nor was there any noticeable asymmetry of the cord.

The dura was sutured with a continuous fine catgut. The remainder of the wound was closed by layer sutures. No drainage was used.

Healing was by primary union.

Six days after operation the pains had disappeared from the right leg but there had been no objective change in the sensory disturbances.

Fourteen days after operation he sat up for two hours, was free from pain and felt generally well. The skin over the right heel had become much more nearly normal and the ulcerated surfaces had nearly healed.

Twenty days after operation, on April 14, 1913, which was just a month after he came into the hospital using crutches because he could not put his right foot to the ground, he left the hospital carrying his crutches under his arm and walking with only a slight limp. His general condition was excellent.

Dr. Cornwell, consulting neurologist of the hospital, examined him the day of his departure from hospital and left this note: "The patient shows practically complete restoration of sensory function from the external malleolus to the knee. There still remains a considerable loss of sensation to pain between the external malleolus and the plantar surface of the heel. There is also a considerable, though not complete, loss of pain sense along the course of the sciatic nerve from its pelvic exit to a point just above the popliteal space. The trophic disturbances in the foot have improved very materially, the only objective signs left being a moderate edema of the foot and a lower surface temperature as compared to the other foot. He still walks with a slight limp but without either crutches or cane which before had been indispensable."

March 24, 1914, one year after operation the sensory conditions remained exactly the same as when he left the hospital. Four weeks after leaving the hospital, which was seven weeks after operation, he took charge of the stock room and wagon loading department of a wholesale drug concern. He has never missed a day out of the whole year. He often shovels several tons of heavy fertilizing mixtures into carts in the course of the day's work, and frequently is called upon to lift 200 lbs. at a time. He walks long distances. The right leg gives him absolutely no trouble. The trophic disturbance on the outer side of the foot has never recurred, in spite of his hard work and the fact that he wears very coarse rough shoes.

His post operative state should be contrasted with the fact that for three months before operation he could not put his foot to the ground, and for four months preceding that, i. e., seven months in all, he had been unable to work because of the pain caused by muscular effort.

These cases have many features in common and are almost counterparts of the case reported by Krause. In three cases there was a history of exposure, twice to inclement weather and once to street car injury. In each instance the onset was in the form of severe neuralgic root pains referred to a fairly definite cord level. After these pains, which underwent remissions and exacerbations, had lasted for a variable length of time, there came a more severe attack of pain which was soon followed by motor disturbances, loss of sphincteric control and the other phenomena indicative of a transverse lesion of the cord. In the fourth case there were no motor nor sphincteric disabilities so that in this case the diagnosis will be questioned, but in every particular other than these it fits so well into the series that it seemed worth while to include it.

In each case the upper level of the lesion was pretty clearly indicated.

In two of the four there were trophic disturbances, one a bed-sore over the end of the spine, the other a disturbance over the outer side of the heel and foot.

In the two cases in which a temperature record has been preserved one ran up to 102° F., and the other to 103° F. during the attack.

The interval between the onset of definite symptoms and operative relief was Case 1, thirteen months; Case 2, three months; Case 3, seven months; while Case 4 had had seven months of disabling pain although his street car trauma which had occurred three years before had been followed by more or less continual discomfort.

In each case the cord was exposed by a unilateral laminectomy. In none of these cases was there any evidence of bone disease. The cord was very much congested and was obviously swollen but not sufficiently to fill out the dural canal. In Case 2 the swelling was more evident in the right half of the cord though there was no circumscribed tumefaction. An incision was made in the post-

erior column of the right side. In Case 4, the symptoms were unilateral, the cord was equally congested on both sides.

In Case 3, in addition to the congenital narrowing of the bony canal which greatly aggravated the pressure effects, there was distinct inflammatory swelling of the dura as well as of the cord. The effect of these combined factors was to eliminate the flow of spinal fluid past this portion of the cord and to cause a cessation of circulatory pulsation in the cord. Following decompression with wide opening of the dura and longitudinal incision of the posterior column of the cord there was very soon a relief of tension as indicated by the return of pulsation in the cord.

With the clinical histories and operative findings the diagnosis of meningomyelitis is established apparently in all the series with the possible exception of the fourth which was somewhat atypical.

In none of the series was there any postoperative shock, or any exacerbation of the symptoms even of a temporary nature.

In Case 1 operation had no effect whatever and there was a steady progression of the symptoms until death occurred on the 24th day after operation. Unfortunately no autopsy was permitted.

In the remainder of the series the improvement was prompt and remarkable when it is considered that there had been progressive disability over a period of months preceding the operation. Within four to eleven days the pains and objective sensory disturbances had largely disappeared, the sphincteric control had returned shortly after that, and motor power had reappeared in eight days in Case 3, although he had been bedridden and unable to move his legs for months. In sixteen days he was walking the length of the ward.

The last two of the series are still under observation. They have suffered no setbacks since operation, have no pains and no motor disturbances, and may fairly be considered cured.

The second case remained under observation only about five weeks, but during that time sphincteric control had returned, the sensory symptoms had nearly disappeared, and the return of voluntary power in the muscles of the lower extremities was well advanced, so that he also should be classed as a successful result. Rumor says that he died several months later but nothing can be learned definitely as to the fact or of the cause of death.

Statements as to the way in which decompression acts must

of course be speculative. To us the most rational explanation lies in an improvement in the local circulation with the resulting more rapid absorption of the inflammatory infiltration and the return of function in the damaged areas of cord. This is especially corroborated by the findings and results in the third case. Here the combination of congenital narrowing of the bony canal, with marked inflammatory thickening of the dura and swelling of the cord substance had caused pulsation to cease. Decompression, free splitting of the dura, and incision of the posterior column of the cord caused, within very few minutes, the return of full pulsation in the cord substance. Cord function returned in a surprisingly short interval.

Just what value lies in the incision of the posterior columns of the cord remains to be proven by further investigation, but it would seem to improve the drainage of the inflammatory exudate away from the cord into the spinal fluid and so hasten the return of the cord to normal.

The experimental work on the traumatized cords of dogs, reported before this association by Allen of Philadelphia, in which free incision of the posterior columns greatly lessened the amount of permanent damage from a given degree of injury to the cord, strongly argues for the addition of this detail of technic to the bony decompression and free division of the dura.

Properly executed incision into the posterior columns of the cord, in the living human subject, has repeatedly been shown to be innocuous by Elsberg, and others as well as myself.

CONCLUSIONS

1. Selected cases of meningomyelitis are susceptible to surgical treatment, where the findings indicate the segmental level.
2. This treatment should consist in laminectomy, free opening of the dura, and, probably, incision into the posterior columns of the cord, especially in those cases showing marked infiltration and swelling of the cord.
3. This operation, properly performed, adds very little to the jeopardy of the patient, but, on the contrary, seems to greatly diminish the period of convalescence and to lead to a more nearly normal return of function in the cord than is usual in these cases when treated expectantly. Many surgeons have demonstrated that laminectomy is not a very dangerous operation.

4. Decompression probably acts favorably by causing freer circulation, with the more rapid absorption of the inflammatory exudate. Incision of the cord probably facilitates drainage.

5. The small risk inherent in laminectomy, and the great advantage likely to accrue to the patient, argue much in favor of the surgical treatment of these cases. The same facts also argue for the earlier employment of exploratory laminectomy in most spinal cases where the diagnosis is not perfectly clear, but where the symptoms point to some cord level as the seat of trouble.

THE PATHOLOGY OF TABETIC OCULAR PALSY WITH
REMARKS ON THE RELATION OF SYPHILIS
TO THE SO-CALLED PARASYPHILITIC
DISEASES¹

BY WILLIAM G. SPILLER, M.D.

PROFESSOR OF NEUROPATHOLOGY IN THE UNIVERSITY OF PENNSYLVANIA

The long struggle, led especially by Fournier and Erb, to establish a relationship between syphilis and the diseases known under the term of parasyphilis has been crowned with success, but since the finding of the *Spirochæta pallida* in paresis and tabes by Noguchi and Moore, and the confirmation of these findings by other investigators, the parasyphilitic diseases have become the subject of an important discussion. Our present knowledge of syphilis of the nervous system has been presented clearly in the recent papers by Nonne at the meetings of the International Medical Congress and the Deutsche Naturforscher und Aerzte. We recognize tabes, paresis, tabo-paresis, primary optic atrophy and cerebro-spinal syphilis as caused by the *Spirochæta pallida*, and these disorders are distinct clinical forms, but many clinical and pathological cases make us consider whether the sharp distinctions are invariably to be maintained, whether tabes and paresis are truly parasyphilitic diseases or are more closely syphilitic affections. I have frequently had difficulty in deciding whether a certain clinical case should be regarded as one of tabes or spinal syphilis, and while paresis and cerebral syphilis are comparatively sharp clinical forms, there are cases where one may hesitate to decide whether the diagnosis should be paresis or cerebral syphilis, and cases even have been described in which cerebral syphilis was supposed to be associated in the same person with paresis. There is reason to believe that special strains of the *Spirochæta pallida* may exist, as shown by Henry J. Nichols,² and may cause different syphilitic diseases of the nervous system.

The paper by Nonne³ on the present status of the syphilis tabes

¹ Read at the fortieth annual meeting of the American Neurological Association, May 7, 8 and 9, 1914.

² Nichols, Journal of Experimental Medicine, April 1, 1914, p. 362.

³ Nonne, Deutsche Zeitschrift für Nervenheilkunde, Vol. 49, p. 384.

paresis question is so important, and apparently so unknown to some, that it seems well to give a brief abstract of it.

Erb gradually became convinced of the true syphilitic nature of the so-called parasyphilitic diseases, and doubted because of many clinical facts whether one could speak of metasyphilitic, parasyphilitic, or post-syphilitic diseases. Hirschl regarded paresis as nothing more than a late effect of syphilis, as a syphilitic encephalitis with termination in syphilitic cerebral atrophy. Raymond took the same stand in the discussion held in Paris over paresis, and Leredde before 1908 declared that tabes and paresis are anatomical and clinical forms of nervous syphilis, the results of a slowly developing syphilitic process. Many transitional forms occur between paresis and tabes on one side and the "true" syphilitic diseases on the other. The examinations of the cerebro-spinal fluid have given the same results for tabes and paresis as for nervous syphilis, except that there are quantitative but not qualitative differences. Jakob and Kafka have shown that paresis may be very typical in its clinical serologic appearances, and that what appears to be typical paresis is shown not to be paresis by the microscope. This has been demonstrated also by Schoenhals, and Nissl emphasizes this fact. Everyone knows, says Nonne, of cases in which the clinical picture at first was that of arterial or meningeal cerebral syphilis but later was typically that of paresis. He has seen such cases almost every year. As for a difference regarding the time interval there are many cases in the literature in which paresis and tabes have appeared within two or three years after the infection. Nonne has seen tabes begin one and a half years after the infection.

As for the effect of treatment, everyone knows that antisyphilitic treatment fails in the arteritic or endarteritic form of syphilis of the brain and cord. Erb and Nonne declare that antisyphilitic treatment is of service in tabes, and others assert the same for paresis.

Nonne says it is certain that the characteristics of paresis as described by Nissl and Alzheimer exist, but Nissl himself acknowledges that all the lesions of paresis found in the cells, vessels, and neuroglia, and the appearance of plasma cells occur with other processes, and the only essential histological features of paresis are the extent and intensity of the vascular changes, the great extent of the alteration of cells, the intense loss of fibers, the situa-

tion of the neuroglial proliferation, the disturbance of the cell arrangement, the preference of plasma cells and lymphocytes for the sheaths of the vessels, and the greater implication of the frontal lobes. Obersteiner, however, has shown that paresis affects the whole brain, the white matter as well as the gray, the cerebellum and spinal cord, and Nonne says all the findings of paresis in their individuality present no differences from those of other syphilitic diseases. Binswanger long ago described the combination of paresis and true syphilis of the brain, and this combination has been shown by Alzheimer, Sträussler and A. Jakobs, and such cases are becoming common.

Tabes formerly was believed to be a primary degeneration of the nervous system. Dejerine and later Dinkler described meningitis occurring with tabes, but this was supposed to be merely a combination of lesions. Schröder in 1906 and E. Meyer and Gustav Oppenheim in 1908 showed that collections of lymphocytes and plasma cells in the perivascular lymph sheaths occur in tabes as in paresis in the pia and the vessels in the pial septa, and even within the posterior and lateral columns of the cord, therefore says Nonne we must reconsider whether tabes is primarily a disease of the parenchyma of the spinal cord.

The most characteristic lesion of cerebro-spinal syphilis, in my experience, is round cell infiltration, lymphocytic infiltration, of the pia. It is often associated with perivascular round cell infiltration within the brain and cord substance. It is not pathognomonic and may occur with some other conditions, but I believe there is no acute manifestation of syphilis of the cerebro-spinal axis without it. Changes in the walls of the blood vessels, thickening of the intima, often are present, but the lymphocytic infiltration may be the only lesion. I have a brain in my possession that shows a moderate but distinct lymphocytic infiltration of the pia and no pronounced change in the vessels. It was from a patient who had been under the care of Dr. James Tyson and Dr. William F. Norris and had had clinical signs of syphilis thirty-one years, and had received much anti-syphilitic treatment. I have other specimens justifying the statement that the lymphocytic infiltration may be the only lesion of syphilis.

It is questionable whether these lymphocytic cells act mechanically by pressure; where they are in large masses they probably exert pressure on nerve fibers passing through the masses. The

lymphocytic infiltration has appeared to me as an index of the intensity of the pathological condition, as a reaction to a toxic condition of the cerebro-spinal fluid, and it is this toxicity which produces degeneration of nerve fibers and the lymphocytic infiltration; therefore the lymphocytic infiltration always has a pathological value.

We see clinical cases of so-called monosymptomatic tabes; or cases in which two or three signs of tabes are present. Some times microscopical examination shows a pronounced, but not necessarily intense, lymphocytic infiltration of the pia and slight degeneration of the posterior columns as in tabes. Is this incipient tabes? Why not equally well incipient syphilis? The lesions of syphilis may not develop beyond a lymphocytic infiltration of the pia in thirty-one years, as shown by the case referred to above, and if some degeneration of the posterior roots occurs in such a case the process is not essentially different.

Difference of opinion exists as to the nuclear or nerve origin of tabetic ocular palsies and a distinction is made by some in the pathology of the ocular palsies of tabes and brain syphilis. It would seem from the study of the following case of complete bilateral tabetic ophthalmoplegia that this distinction is unreliable and that in this as well as in other respects the sharp limitation formerly made of parasyphilis and cerebro-spinal syphilis needs much modification.

J. F. laboratory no. 631 was admitted to my service at the Philadelphia General Hospital Jan. 29, 1913, and died Feb. 19, 1913. He had a chancre in 1895. The right knee became much swollen in the winter of 1908 and the swelling was without spontaneous pain or pain on motion. About 1911 the right eye closed and within a few months the left eye closed so that to see he was obliged to raise either upper lid with his fingers. He had sharp shooting pains in the lower limbs before the arthropathy of the right knee developed, and his lower limbs felt as though they were pricked with pins. He complained of girdle sensation and said he felt as though a strap were tightened about the lower ribs and waist. In walking on the street he would stub his toes thinking he had raised his toes high enough to clear the ground. He evidently had at this time impairment of the sense of position. His urine dribbled occasionally and at times urination was accomplished by much straining.

When he came into the hospital the right knee was greatly swollen and crepitation was obtained upon pressure. The right leg could be rotated outwardly and inwardly to an abnormal de-

gree. The left lower limb also presented much hypotonia. The lower limbs were much atrophied. The tendon reflexes of the upper limbs were diminished. The patellar and Achilles tendon reflexes were lost. The finger to nose test showed ataxia. Dr. Hansell examined the eyes Feb. 2, 1913, and reported that there was total internal and external ophthalmoplegia of both eyes, with paralytic divergence. The iridic reaction was lost. The optic nerves were partially degenerated.

At the necropsy performed by Dr. G. McConnell, the enlarged right knee joint was somewhat fluctuating and hard irregular masses could be felt within it. The upper portion of the tibia was enlarged and roughened and the knee joint was so damaged that the leg could be moved in all directions. When this joint was opened about 500 cc. of thick brownish fluid escaped, leaving a sac lined with a thick membrane from which hung numerous irregular calcified masses. The lower end of the femur was thickened and very irregular, but appeared to be covered by the periosteum.

The region of the ocular nerves in the brainstem was cut in microscopical serial sections and one of every ten sections was stained by the Weigert hematoxylin method. A similar series was made with nuclear stains, and Nissl sections also were made.

Much lymphocytic infiltration was found in the pia of the spinal cord and base of the brain and the basilar artery presented much endarteritis. The posterior lumbar roots and the posterior columns of the cord were degenerated in the usual manner of tabes.

The ocular nerves were cut separately from the brain. Each oculomotor nerve showed much lymphocytic infiltration and this infiltration was more intense in the left nerve. These nerves were much and equally degenerated, and the degeneration was greater than that of the abducent and trochlear nerves. The oculomotor nuclei were greatly atrophied and contained few nerve cells, and the oculomotor fibers within the cerebral peduncles were much fewer than normal. Patches of rarefied tissue containing few nerve fibers were found within the oculomotor nuclei. The Edinger-Westphal nuclei were much better preserved than the other portions of the oculomotor nuclei.

The trochlear nerves showed slight lymphocytic infiltration.

The left abducent nucleus was considerably more atrophied than the right although the right nucleus was much affected. The abducent nerves were much atrophied, the left more so than the right. Both these nerves presented considerable lymphocytic infiltration, and the left more than the right.

The degeneration of the oculomotor nerves was so far advanced that it was impossible to decide whether the degeneration began in the nerve or in the nucleus, but the findings in the abducent nerves were of importance in this respect. The left nucleus and nerve

were considerably more degenerated than the right, and the lymphocytic infiltration of the left nerve at its exit from the upper medulla oblongata was greater than that of the right. This would seem to indicate that the syphilitic process was primarily in the nerve rather than the nucleus, and would make a distinction between the ocular palsies of tabes and syphilis unreliable. This case clinically was distinctly one of tabes, and the pathological findings in the cord were those of tabes.

Among writers who have given attention to the pathology of ocular palsies in tabes and syphilis the view has been largely held that tabetic palsies are usually of nuclear origin while those of brain syphilis are usually from nerve degeneration. Thus Uthoff gives a long list of authors as references for his statement that tabetic ocular palsies are often nuclear in origin, but he states that many of these authors speak of degeneration of root fibers of these ocular nerves and of the nerves themselves in addition to the nuclear lesions as almost constant findings. Cases of tabetic ocular palsy in which only the nuclei of these nerves are degenerated are very rare, chiefly because the examination is made long after the onset of the palsy when the nerve lesions must follow the nuclear. In some cases the cells are reported as more degenerated than the nerve fibers of the ocular nerves, and on this account the nuclear degeneration is supposed to be primary. In other cases the nerve lesions are more intense than the nuclear. Nevertheless Uthoff asserts that a considerable number of cases show the degeneration to be primarily nuclear either by absence of peripheral change or greater degeneration of the nuclei, and he believes these cases are the more numerous. Ependymal proliferation of the aqueduct of Sylvius and the fourth ventricle appears to be relatively seldom the cause of tabetic ocular palsy. Hemorrhages in the ocular nuclei are seldom observed and in part at least occur shortly before death.

Uthoff⁴ states that pronounced external or total ophthalmoplegia has occurred only in about two per cent. of his tabes cases, but he found about 40 cases with necropsy in the literature.

Uthoff in contrast to his views regarding the greater frequency of nuclear lesions as the cause of tabetic ocular palsy, states that he believes single or double oculomotor palsy in syphilis, even when other symptoms fail, very rarely is of nuclear origin and almost always is basal.

⁴ Uthoff, Graefe-Saemisch Handbuch der gesamten Augenheilkunde, Vol. XI, part 2A.

He refers to a case reported by v. Monakow of total ophthalmoplegia in which the oculomotor nuclei were degenerated and the abducens and trochlearis nuclei were almost normal. He believed that the ophthalmoplegia in part was caused by the implication of fibers from the cerebral cortex to the nuclei.

Nonne⁵ makes a distinction between the ocular palsies of tabes and syphilis. The disappearance of ocular palsy is not characteristic of syphilis, he says, as this very often occurs in post-syphilitic tabes.

He refers to some cases in which the lesions were at the sphenoidal fissure, in these cases the symptoms were amaurosis, paralysis of all external and internal ocular muscles, anesthesia of the first branch of the trigeminus, pain in the depth of the orbit, edema of the upper lid and slight exophthalmos.

He states that although one might expect to find isolated nuclear disease in syphilitic ocular palsy, as individual branches of the oculomotorius are often paralyzed, no necropsy has shown this. Wilbrand and Saenger, he says, make the same statement.

Wilbrand and Saenger⁶ state that in the majority of cases of tabetic ophthalmoplegia intense atrophy of the nuclei of the ocular nerves was found as well as of the nerve roots, nerves and muscles. They speak of the lesion as primarily nuclear (p. 153).

In speaking of Hutchinson's case of ophthalmoplegia examined microscopically by Gowers, these authors say syphilis as the cause of primary nuclear degeneration in this case is doubtful and the case must be regarded as one of tabes. Oppenheim's case, they state, is the only one they know of in which primary nuclear atrophy occurred during the period of constitutional syphilis (p. 333).

They thus sharply define tabetic ophthalmoplegia as primarily nuclear in the majority of cases, and syphilitic ophthalmoplegia as primarily basal.

The question arises as to the influence of the ocular centers upon one another. That some sort of connection must exist is shown by the paralysis of lateral associated movement with preservation of the function of the internal rectus in convergence. Leonard J. Kidd⁷ has recently discussed the method of connection of these ocular muscles. He shows that the widely accepted teach-

⁵ Nonne, *Syphilis und Nervensystem*. Second edition.

⁶ Wilbrand und Saenger, *Die Neurologie des Auges*, Vol. I.

⁷ Kidd, *Review of Neurology and Psychiatry*, Oct., 1913, p. 507.

ing that lateral conjugate eye-movements are carried out by virtue of fibers, which the sixth nucleus is alleged to send to the contralateral third nucleus or root for the innervation of the internal rectus muscle, has been demonstrated to be erroneous by the sure test of carefully planned and executed experimentation. He calls this sixth nucleus hypothesis the Duval-Laborde hypothesis.

Van der Schueren has established that in the rabbit the posterior longitudinal bundle in its course between the level of the sixth nucleus and the fourth and third nerves contains ascending fibers which are connected with the cells of the homolateral fourth and third nuclei. The sixth nucleus sends no fibers into the posterior longitudinal bundle, and no neurones connect the sixth nucleus with the fourth and third nuclei. Yet in all cases of loss of lateral conjugate eye movements, accompanied by preservation of the action of the contralateral internal rectus muscle in convergence, which have been carefully examined pathologically, Kidd says evidence is found of the presence of degenerated fibers in the posterior longitudinal bundle. Van Gehuchten expressed the opinion in 1892 that the posterior longitudinal bundle seems to be sufficient to carry out the lateral conjugate eye movements by means of the fibers which pass in that bundle to the various ocular nuclei. Many other authors have held the same opinion, and Kidd thinks we can have no reasonable doubt that this opinion is correct, and that the infracortical mechanism for the lateral conjugate eye movements depends on the ascending vestibulo-ocular fibers of the posterior longitudinal bundle.

If this be the correct explanation it is hard to understand how a lesion of nerve fibers of one ocular nerve could affect either of the other two ocular nerves. Why then do we get complete ophthalmoplegia in tabes or brain syphilis.

I have seen bilateral facial palsy in syphilis without any other symptoms and without any other nerve involvement, and the syphilitic meningitis must have picked out these two nerves alone. It is therefore possible that the ocular nerves should be paralyzed alone. Whether they are more susceptible to the syphilitic poison than are other cranial nerves is questionable. A more probable explanation, it seems to me, is to be found in the syphilitic meningitis which is most pronounced about the cerebral peduncles. Here the ocular nerves are near one another. The third nerve arises on the inner side of the peduncle, the fourth nerve passes on

the outer side of the peduncle, and the sixth nerve takes its course directly forward on the pons from its exit at the junction of the pons with the medulla oblongata, and comes close to the cerebral peduncle. We can readily understand why all the ocular nerves should be paralyzed in tabes or cerebral syphilis from meningitis about the cerebral peduncles, without implication of other cranial nerves, while such selective palsy would be difficult to explain on a nuclear hypothesis.

I have examined the sections from eleven cases diagnosed clinically as tabes. In all except two of these there was considerable lymphocytic infiltration of the pia and in some there was thickening of the pial vessels. In the two cases referred to above the lymphocytic infiltration was slight, and in one of these the degeneration of the posterior columns was slight; it was incipient tabes. I could add to this number, but eleven cases seem to be sufficient to permit an opinion.

The question of lymphocytic infiltration has been studied by Bresowsky.⁸ He examined the spinal cords from 40 cases of tabes and in all, old as well as recent cases, meningitis was found, and was of severe type in about half of the cases. He believes the tabetic degeneration depends on the meningitis which affects nervous tissue in lessened vitality and this brings about the degeneration. It would seem from such findings as these that sharp distinctions pathologically can not always be made between tabes and spinal syphilis, and the differentiation from a clinical aspect may be equally difficult. It is becoming more and more impossible to separate sharply the parasyphilitic diseases of brain and spinal cord from cerebro-spinal syphilis. I believe if careful microscopical examination be made of the region of the origin of the ocular nerves in the brain stem in cases of tabes with ophthalmoplegia, with the attempt to determine whether the lesion be primarily within the nerves or their nuclei, it will be found impossible to regard tabetic palsies of ocular muscles as primarily nuclear in origin. Most cases come to necropsy so late that the nerve fibers and nuclei are both intensely altered. In some instances, as in the case reported in this paper, the degeneration may be found to be greater where the lymphocytic infiltration is more pronounced. I⁹ have discussed the ocular palsies occurring in cerebral syphilis in another paper.

⁸ Bresowsky, *Obersteiner's Arbeiten*, Vol. XX, 1913.

⁹ Spiller, *Van Gehuchten' Festschrift. Le Névraxe*, vol. XIV, 1913.

A CASE OF PROBABLE ENCEPHALITIS DUE TO THE INHALATION OF THE FUMES OF GASOLINE¹

BY CHAS. S. POTTS, M.D.

PROFESSOR OF NEUROLOGY IN THE MEDICO-CHIRURGICAL COLLEGE OF PHILADELPHIA; NEUROLOGIST TO THE PHILADELPHIA GENERAL HOSPITAL

The production of more or less transient toxic symptoms such as headache, nausea, delirium and loss of consciousness by inhaling the fumes of gasoline is not uncommon. The production of more permanent symptoms referable to the nervous system is apparently not very common when the few cases of such that have been reported and the large quantities of gasoline now used are considered. Gasoline as sold contains a number of the different products of petrol distillation, such as naphtha, benzine and petroleum ether. The amount of these different hydrocarbons in commercial gasoline is not fixed, but differs in that of different manufacturers. D. Felix² in 1872 studied the effects of the inhalation of both benzine and petroleum ether and found that each caused vertigo, nausea, sleepiness, injection of the conjunctiva, burning pains in the chest and irritating cough. If the inhalation was continued loss of consciousness resulted. Oliver³ speaks of the inhalation of petrol causing headache, vertigo, vomiting and unconsciousness. He further says⁴ that inhalation of either benzine or naphtha will cause mental depression, hallucinations, loss of memory, blindness and multiple neuritis. Rambosek⁵ says that some naphtha products irritate the respiratory organs while others affect the *central nervous system*. The same author⁶ states that poisoning may occur from cleaning out petroleum stills and mixing vessels and in emptying out residues, and occasionally from the use of benzine in chemical cleaning. He also speaks of the possibility of poisoning in the refining of naphtha from the inhalation of the vapor of the light oils, benzine and gasoline. Fatal cases have been recorded in badly ventilated work rooms in which

¹ Patient shown to the Philadelphia Neurological Society. March 27, 1914. Read by title at the fortieth annual meeting of the American Neurological Association, May 7, 8 and 9, 1914.

the products of distillation are collected. He mentions the case of a workman who had entered an open benzine still to scrape the walls free of crusts containing benzine. He was found unconscious and died some hours later. He mentions another case of a man who started to clean a benzine still. When he entered the opening he became unconscious and cyanosed, the pulse was weak, the legs were spastic and the reflexes absent. These symptoms disappeared ninety minutes later. Another man who examined the outlet of a petroleum tank had symptoms in two minutes. Gowers⁷ (quoted by Rambousek) reports a case of chronic poisoning following the frequent inhalation of gases given off from a petroleum motor. The symptoms were slurring speech, difficulty in swallowing, and weakness of the facial muscles. The symptoms disappeared when the man ceased his occupation but returned when he resumed it. Gowers believed the symptoms to be due to petroleum gas poisoning. In this connection it may be stated that among the waste products given off by a gasoline motor is carbon monoxide, such motors are therefore dangerous when running in a closed room. A case is reported⁸ in which two men who were in a garage with closed windows while the motor was running were found unconscious. One in addition was cyanosed, the pulse weak and rapid, the other was pale and had clonic spasms. Both recovered. The possibility of the conditions described by Dana,⁹ Spiller and McConnell¹⁰ and others occurring should be remembered when such exposure has occurred.

The case I report was a white male aged 45 years, who was admitted to the Medico-Chirurgical Hospital in an unconscious condition on November 14, 1913. The history obtained at the time was that his occupation had been that of filling the tanks of automobiles with gasoline and while at work he had fallen over unconscious. He remained so for several hours and then passed into a stuporous condition which continued until November 25, when aroused from this he complained of intense headache. On admission his temperature was 97.4° Fahr., the pulse 80 and the respiration 20. A few hours later the temperature was 97° Fahr. and during the following three days dropped to that point several times, the pulse ranging from 72 to 90. When admitted he was regarded as suffering from gasoline poisoning. He was transferred to my service on November 24, and was first seen by me on that date. He was still quite stupid but could be aroused. It was then noticed that there was ptosis of the right eyelid and that the eyeball was drawn downward and to the right. Some

headache was complained of. The next day (November 25) the mental condition had much improved and he was examined by Dr. Weisenburg, an abstract of his findings being as follows:

Nearly complete oculomotor palsy on the right side, the only movements present being a slight contraction of the pupil to light and slight power of raising the eyelid. On the left side the pupil was smaller than that of the right and responded to light, all movements of the eyeball were lost excepting inward rotation and slight rotation outward. The eyeball was turned in when at rest. Associated movement of both eyes to the right was present. The angle of the mouth could not be drawn as well to the left as to the right (facial palsy of central type) and the arm and leg of this side were much weaker than those of the right. The knee jerks were increased, the left the more so, ankle clonus was present on the left side and plantar irritation caused a tendency to dorsal flexion of the toes on the left. There was marked dysmetria on the left side, the finger, in the finger to nose test, usually going beyond the nose, the movement being quick and jerky. *Adiadochokinesis* was marked in the left arm. *Asynergia* of the left hand was well shown. The same conditions were present in the left leg, the heel to knee test being poorly performed, *asynergia* being present and the muscle tone less as shown by the fact that the thigh could be flexed upon the abdomen much more than on the right side. The station was very poor, the tendency being to fall to the left. The gait was very unsteady, of the cerebellar type, the tendency being to go to the left. The left leg was dragged slightly. All other functions of the nervous system were normal. On November 29, he was carefully examined by the writer, and the following additional facts were elicited: His family and previous history contained nothing of note. He had always been a hard worker, drank three or four glasses of beer daily, did not use tobacco and denied venereal disease, the Wassermann test was negative. He had worked at his present occupation for four months. Two months before admission he was seized one evening with intense headache, nausea and vomiting which lasted all night. He went to work in the morning however but since has had more or less severe headache and dizziness. During this period he began to see double, one image being to one side and above the other. The symptoms present were the same as those found on November 25, except that the ptosis of the left eyelid was somewhat less and the movements of the left eyeball while still sluggish, were better, upward and outward movement being the most impaired. Attempts to move the eyeballs caused coarse nystagmoid movements in the direction of the attempted movement. Ankle clonus had disappeared on the left side. The eye grounds were normal. The functions of the viscera were normal. The treatment consisted at first of tinct. gentian co. as a placebo but was followed by iodide of potassium and strychnine.

Improvement gradually took place and by February the only symptoms remaining were impairment of the functions of the left oculomotor nerve, ataxia of the left arm and possibly slight weakness of the left leg. These symptoms persisted and when last seen in April no further improvement had occurred. There would seem to be no doubt that the symptoms were due to a toxemia of some sort and that it was gasoline would seem to be a justifiable conclusion in view of the premonitory symptoms and the absence of exposure to any other form of poison. The question might be raised whether there was either merely a functional disturbance or an actual organic lesion as indicated by the diagnosis of encephalitis. The writer believes the latter to be justified, because while most of the symptoms have disappeared, there still, after a lapse of a number of months, remained evidence of permanent damage to some functions. The writer believes that a lesion in the region of the aqueduct of Sylvius would explain the symptoms.

In the discussion which followed the exhibition of the patient at the Philadelphia Neurological Society, the tendency of acute intoxications to cause cerebellar symptoms was commented upon.

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ON THE DIAGNOSTIC VALUE OF HALLUCINATIONS:
BASED ON A STUDY OF 500 CASES OF MENTAL
DISEASE¹

BY A. WARREN STEARNS, M.D.

ASSISTANT TO THE STATE BOARD OF INSANITY, MASSACHUSETTS; PHYSICIAN
FOR MENTAL DISEASES, BOSTON DISPENSARY; INSTRUCTOR IN NEUROLOGY,
TUFTS MEDICAL SCHOOL

In most state hospitals where the usual form of admission is by regular commitment, the principal medical problem is classification according to form of insanity. At the Psychopathic Hospital (Boston, Massachusetts) the question of whether or not the patient is sane comes first of all, and, added to this, is the matter of classification. Also, it is necessary to decide these two questions in a much shorter period of time than is usually available in hospitals for the insane, as most of the admissions are for temporary care only. This condition has made it very important to seek for symptoms which are easy to detect and of differential value, because the diagnosis rests more upon the interpretation of symptoms than in following the course of the disease. This study has been undertaken with the hope that the occurrence and type of hallucinations might be made of some differential value in diagnosis.

A group of 500 cases taken in alphabetical order has been studied to see first, how many had hallucinations, next, whether they were of hearing, vision or other type, and lastly, to determine whether there were any which seemed especially characteristic of any form of disease. By taking cases in alphabetical order not only all types are included, but all periods of the hospital's work are embraced, thus excluding the influence of any particular diagnostician. It is obvious that there is no way of proving diagnoses or the presence or absence of hallucinations, so the records have been taken literally; thus any error is, at least, evenly distributed.

¹ Being Contributions from the Psychopathic Hospital, Boston, Massachusetts, Number 63 (1914. 29), read at meeting of the Norfolk District Medical Society, March 31, 1914.

Table I shows the number of patients with each disease, the number of each having hallucinations and the type of these hallucinations when they occurred. Table II shows the same given in percentages.

TABLE I
SHOWING NUMBER HALLUCINATED, AND TYPE IN EACH DISEASE

Diagnosis.	No.	Present.	Absent.	Auditory.	Visual.	Other.
Unclassified.....	108	29	79	22	9	0
Dementia præcox.....	105	74	31	71	22	10
Not insane.....	65	4	61	2	2	0
Manic-depressive insanity.....	54	10	44	7	5	0
General paralysis.....	37	10	27	9	4	2
Alcoholic hallucinosis.....	31	31	0	30	12	4
Organic dementia-arteriosclerosis....	24	2	22	2	2	0
Senile dementia.....	15	1	14	0	1	0
Delirium tremens.....	14	14	0	11	12	2
Korsakow's psychosis.....	14	3	11	2	2	0
Epileptic psychosis.....	10	3	7	1	2	0
Morphine psychosis.....	6	5	1	3	2	0
Miscellaneous psychosis.....	6	3	3	3	0	1
Toxic psychosis.....	5	2	3	1	1	0
Cerebral syphilis.....	4	1	3	1	1	0
Brain tumor.....	2	1	1	1	0	0
Total.....	500	193	307	166	77	19
Total in per cent.....		38.6	61.4	33.2	15.4	3.8

TABLE II
SHOWING SAME AS TABLE I IN PERCENTAGES

	Present.	Absent.	Auditory.	Visual.	Other.
Alcoholic hallucinosis.....	100	0	97	4	13
Delirium tremens.....	100	0	78	85	14
Morphine psychosis.....	83	17	50	34	0
Dementia præcox.....	70	30	68	21	10
Miscellaneous.....	50	50	50	0	17
Brain tumor.....	50	50	50	0	0
Toxic psychosis.....	40	60	20	20	0
Epileptic psychosis.....	30	70	10	10	20
Unclassified.....	27	73	20	8	0
General paralysis.....	27	73	24	11	5
Cerebral syphilis.....	25	75	25	25	0
Korsakow's psychosis.....	21	79	14	14	0
Manic-depressive insanity.....	18	82	13	9	0
Organic dementia-arteriosclerosis.....	8	92	8	8	0
Senile dementia.....	7	93	0	7	0
Not insane.....	6	94	3	3	0

If we take up the various diseases in order of frequency of hallucinations, Alcoholic Hallucinosis comes first. Of the 31 cases, 100 per cent. were hallucinated, 97 with auditory, 4 with visual, and 13 with other types. As the presence of visual hallucinations has been used as pointing towards the diagnosis of delirium tremens and of auditory toward alcoholic hallucinosis, these figures might be expected. Examination of the delirium tremens group shows that 100 per cent. of the 14 cases were hallucinated, 78 auditory, 85 visual and 14 other types. It is somewhat of a surprise to note the large number of this group who had auditory hallucinations. The frequency of clouding of consciousness in delirium tremens may explain the presence of visual hallucinations in this disease. Auditory hallucinations are about equally prevalent in both and given a clouding of consciousness, illusions would be more apt to occur so changing the clinical picture. This makes possible a previous contention that these two diseases are usually one and the same process when of short duration and following a debauch.¹

The type of hallucinations in these two diseases are familiar to all, but it seems true that threatening accusing voices are common in both. Attention has also been called to the frequent occurrence of hallucinations of taste, smell and touch.

The morphine group is very similar, the one case not showing hallucinations had recovered when she reached the hospital and though delirious outside, she had no memory of her symptoms. The type was much like the alcoholic.

The dementia præcox group comes next. Of 105 cases, 70 per cent. were hallucinated, 68 auditory, 21 visual and 10 other types. In the 31 cases not showing hallucinations, 11, though they denied their presence, showed some evidence of their existence, 3 were catatonic and did not speak, 6 could not be examined because of violence or language difficulty and in only 11 cases, or 10 per cent., could it be fairly said that they were absent. This bears out the statement of Bleuler that "almost every schizophrenic in institutions hears voices."¹ Abusive and threatening voices are common to all types of this disease and when alcohol can be excluded are very characteristic. Visual, especially what Dr. Southard has called "scenic" hallucinations, are more common in the catatonic

¹ Boston Med. & Surg. Journ., Vol. 12, Sept. 18, 1913.

form, while single words, meaningless sentences and impersonal conversation occur most often in the hebephrenic form.

The miscellaneous group consists of diseases occurring but once so is not available for statistical purposes. Of the 2 brain tumor cases one thought he heard the voice of a friend calling and this may have been an illusion. Of the 5 toxic cases, 2 had indefinite hallucinations which were not unusual. Of the 3 epileptics who had hallucinations, 1 had visual, 1 tactile and 1 gustatory. Twenty-seven per cent. of the unclassified cases were hallucinated, but there seems to be nothing to be gained by analysis of these.

Twenty-five per cent. of the general paralytics were hallucinated, 24 of auditory and 11 of visual type. No two of these were alike and in but 2 cases were hallucinations a prominent symptom. Only one out of four cases of cerebral syphilis gave a history of hallucinations and this one was rather indefinite. In Korsakow's disease, 27 per cent. admitted hallucinations, these usually occurring in a delirious onset.

In the manic-depressive group, but 18 per cent. were hallucinated. Thirteen per cent. of these were auditory and 9 visual, and in 54 cases but 10 had hallucinations. In none of these were they a prominent symptom and in many there is reason to doubt their existence. Seven of these 10 were manic and 3 depressed. A summary of each follows.

No. 1. Elated, said people had talked about her and that she had seen rats in her room at the hospital.

No. 2. Claimed to have seen his employer in the hospital.

No. 3. Elated and euphoric, told of wonderful vision which she had seen.

No. 4. Elated, told of beautiful vision.

No. 5. Elated, talked and argued with imaginary companion.

No. 6. Elated and hyperreligious, claimed to have had communication from God.

No. 7. Elated, told of beautiful bird she had seen.

No. 8. Depressed, thought family had talked about her.

No. 9. Depressed, jumped out a window and afterwards said a voice had said, "Why don't you jump?"

No. 10. Depressed, thought she heard her family laughing at her.

It seems quite possible that these supposed hallucinations in the manic cases are but expressions of their elation and plays of fancy,

and that those of the depressed cases are but illusions due to their anxious state of mind. The infrequency of hallucinations is often mentioned in the literature. Remond, referring to mania, says, "Rarer still than delusions are hallucinations. . . ."² De-Fursac, "Hallucinations are rare and fleeting."³ Diefendorf, "Hallucinations are rare, except in the delirious form of the manic phase and in the more marked stuporous depressions, but even here they are neither a prominent nor persistent feature. Furthermore, the hallucinations do not have the same sensory distinctness common to the sense deceptions of dementia præcox."⁴ G. Deny and Paul Camus, "The existence of true hallucinations in the course of depressed states is a rare phenomenon. There are more often illusions and false interpretations of real sensation." And in the manic states, they speak of hallucinations and illusions as, "rare and usually ephemeral." Also, "Illusions are more common, especially illusions of memory."⁵

Kraepelin⁶ states that false perceptions are not often in the foreground of the manic-depressive picture, but that they do occur in many different forms of the disease. These false perceptions are illusions rather than hallucinations and are favored by the imperfect fleeting perceptions and lively emotions of the patient, and in point of fact always correspond to his thoughts and mood.

True hallucinations also occur, especially by night though also by day, as a rule somewhat startling visions of faces and various forms and objects in the midst of whatever patient is looking at (e. g., a wall or door), or also auditory, somewhat ill-defined and fragmentary statements suitable to the mood of the patient (who can rarely repeat the exact words, as might a victim of dementia præcox or alcoholic hallucinosis).

Stransky⁷ states in his monograph on Manic-Depressive Insanity that although hallucinations occur in the exalted phases of manic-depressive insanity, yet they do not form a typical symptom thereof, are completely lacking in the majority of cases and hardly dominate the picture, except in those delirious conditions which are counted by many authors as belonging to manic-depressive insanity. On the other hand, Stransky states that hallucinations play a far greater part in the depressive phases of manic-depressive insanity and at times, he believes, in delusional states or in apprehensive agitation which is sometimes found in the depressive phase of manic-depressive insanity. Illusions or hallucinations,

as a rule, in the form of voices, are not infrequent. The content of these voices is either closely akin to the dominant emotional and intellectual state or else it forms a very sharp contrast thereto. In the latter event, these voices are in the form of imperative ideas, as it were, expressed aloud.

Stransky states that hallucinations of irrelevant nature are very rare and perhaps do not occur in cases in which the diagnosis is beyond suspicion.

Ziehen⁸ states that mania, a form of the affective psychoses, exhibits in many cases no disorder in the sensory field, nor is there any remarkable lowering of the threshold of stimuli. He states that perhaps one fifth of these cases exhibited hallucinations, as a rule, of vision, characterized by manifoldness and mobility, with moving animals and landscapes. Gustatory, olfactory and tactile hallucinations occur very infrequently. Illusions are perhaps more frequent than hallucinations and among illusions those of visual type are most frequent.

Ziehen goes on to say that very young patients or very old patients, as well as alcoholic and hysterical persons, exhibit a predisposition to hallucinations and illusions. Ziehen classifies as one form of mania that in which there are numerous hallucinations and terms this form *mania hallucinatoria*.

Of course it remains a question how far Ziehen's mania corresponds with the maniacal form of manic-depressive insanity, according to Kraepelin. Ziehen states that about 90 per cent. of these manias recover, that about 5 per cent. of them die, that about 4 per cent. exhibit a permanent defect of intelligence; that occasional cases are transformed into a disease which he terms *secondary hallucinatory paranoia*, and that a few cases, very especially senile cases, are transformed into cases of *chronic mania*.

Ziehen does not reckon among these manias those which are partial phases of periodic or circular insanity. He states that the post-maniacal occurrence of hallucinations and delusions, *without* underlying intelligence defect, is very rare.

But eight per cent. of the arteriosclerotics, or two out of twenty-four, were hallucinated. One of these was delirious and the other saw people who told him to do things. One of the cases of senile dementia thought he saw a negro on the shelf in his room.

Hallucinations occurred least frequently of all in the not-in-

sane group as only six per cent., four out of sixty-five cases, were so affected. Of these:

No. 1. An hysteric, heard buzzing and thought she heard her name called.

No. 2. An hysteric, heard some one crying, "Stop."

No. 3. A child, saw ghosts at night, probably *pavor nocturnus*.

No. 4. A child, saw frightful and fantastic figures at night, *pavor nocturnus*?

These figures are somewhat at variance with those of Gurney¹⁰. He asked the following question of 27,737 supposedly normal people: "Have you ever, when believing yourself to be completely awake, had a vivid impression of seeing or being touched by a living being or inanimate object, or of hearing a voice, which impression, so far as you can discover, was not due to any external physical cause?" 11.96 per cent. answered in the affirmative, by which he inferred that that per cent. of normal people had had an hallucination. Parish¹¹ objects to the fact that most writers use only morbid conditions in studying hallucinations. Nevertheless there seems to be very little in common between the hallucinations occurring as symptoms of insanity and the false perceptions alluded to above, and actual hallucinations point pretty strongly to mental disease.

The following conclusions may be drawn from this work:

1. The presence of hallucinations is indispensable for the diagnosis of alcoholic hallucinosis or delirium tremens, but the type of hallucinations is not a proper criterion for differentiation between these diseases.

2. The frequency of hallucinations in dementia præcox and their rarity in manic-depressive insanity has a bearing on differential diagnosis (provided that Psychopathic Hospital criteria are sound for these two diseases).

3. There are some grounds for doubting the existence of true hallucinations in manic-depressive insanity.

4. Hallucinations seem to be rare in sane persons, even though they be psychopaths.

1. Bleuler. *Schizophrenie*, p. 78.

2. Remond. *Maladies mentales*, pp. 76 and 192.

3. DeFursac. Trans. by Rosanoff, *Outline of Psychiatry*, pp. 348, 453, 356.

4. Diefendorf. *Clinical Psychiatry*, p. 383.

5. G. Deny and Paul Camus. *La Psychose Maniaque-Depressive*, pp. 40 and 45.
6. Kraepelin. *Psychiatrie*, Vol. III.
7. Stransky. *Die Manisch-Depressive Irresein*, p. 15.
8. Ziehen. *Psychiatrie*, pp. 364-365, and p. 392.
9. Tuttle. *Am. Jour. Ins.*, Jan., 1902.
10. Gurney. *Phantasms of the Living*. Quoted by James and Tuttle.
11. Parish. *Hallucinations and Illusions*, preface.
12. James. *Psychology*.

Society Proceedings

FORTIETH ANNUAL MEETING OF THE AMERICAN NEUROLOGICAL ASSOCIATION

HELD AT ALBANY, N. Y., MAY 7, 8, AND 9, 1914

The President, DR. HENRY HUN, in the Chair

(Continued from page 790, vol. 41)

THE CORTICAL CONNECTIONS OF THE RED NUCLEUS

By LaSalle Archambault, M.D.

Lantern slide demonstration of several cases, illustrating well-marked secondary degeneration of the red nucleus in lesions involving circumscribed areas of the cerebral cortex. The distribution and intensity of the degenerative reaction vary with the topography of the causative lesion. The red nucleus receives fibers from the temporal lobe, the frontal lobe and the rolandic region.

Dr. C. K. Mills thought this paper was most interesting because of its bearing upon certain broad generalizations with regard to the organic basis of the cerebral affective motor apparatus. In his own paper he discussed this subject more fully and therefore would say very little about it here. The red nucleus holds a special position in this cerebral affective or tonectic apparatus, forming the connecting link between this cerebral tonectic, the cerebral voluntary motor, the cerebello-rubro-thalamic and the cerebello-rubro-spinal systems.

Dr. Mills said he had never had the opportunity of seeing a better demonstration or listening to a better paper designed to elucidate some of the important but obscure problems of cerebral structure and function.

Dr. Arthur Schuller, of Vienna, gave a demonstration on the Roentgenology of Epilepsy.

Dr. T. M. T. McKennan, Dr. Geo. C. Johnston and Dr. C. H. Henninger, read a paper with the title: Observations on Epileptics, Chiefly from an X-Ray Standpoint. (*See this Journal, 1914.*)

Dr. Sachs said with reference to the remarks of Professor Schuller, one question occurred to him, whether particularly in case of brain tumor he would be inclined to attach any importance to the evidences through the X-ray picture of the enlargement of blood vessels and slight separation of sutures. Whether there would be enough in that sort of evidence to make it of value in the diagnosis of intracranial tumor. We have seen plates with such changes and there has generally been a division of opinion whether that passed beyond physiological limits.

Dr. McKennan said they had examined fourteen of these cases for sugar tolerance. All of these were in the hospital as it is difficult to examine for sugar tolerance unless one has complete control of the patient.

They found increased sugar tolerance in all fourteen cases. This they considered suggestive of hyperpituitarism.

The special point they endeavored to bring out was the fact of the bony changes at the base of the skull and they studied, very often for hours at a time, the X-ray plates and tried to avoid using their imagination in reaching conclusions. It seemed to them that it may take a good many more observations to determine this matter. Their findings seemed to be very suggestive and the fact of the increased bony deposit in itself seemed to be the significant thing. The amount of bony deposit has not so much importance as the fact that there exists an increase in the amount of bone in the region around the pituitary gland.

Dr. Johnson said in some 200 cases they had never found any of the calcareous deposits which Dr. Schuller had been so fortunate as to find.

PHYSIOLOGICAL CHARACTERISTICS IN INSANITY

By S. D. W. Ludlum, M.D.

An analysis of the physiological symptoms that are seen in insanity, regardless of the psychological diagnosis and an arrangement of the cases, studied according to physiological and biochemical investigations.

The similarity of this arrangement with the syndromes as seen in experimental and pathological work on the ductless glands.

Clinical and laboratory methods of observing treatment.

Dr. Knapp asked Dr. Ludlum whether he took into account the stage of the disease in the cases of dementia præcox.

FLEETING ATTACKS OF MANIC DEPRESSIVE PSYCHOSIS

By Menas S. Gregory, M.D.

The literature dealing with transient attacks of Manic Depressive Psychosis is very meager.

Such short attacks are more frequent than the protracted ones requiring hospital treatment, and may last from a few hours to several days. Quite often the condition is not recognized unless accompanied by accentuating etiological factors, especially that of alcohol, which frequently masks the picture. A great majority of periodic drinkers, and especially so-called "pathological drunkards," are really examples of short attacks of Manic Depressive with alcohol.

Short attacks of mild type unaccompanied by alcohol are frequently mistaken for transitory mental conditions of other character, such as hysteria, psychic epilepsy, etc.

These attacks may be associated by the patient with somatic disturbances, and are frequently regarded by the general practitioner as migraine, neuralgia, nervous dyspepsia, nervous heart, liver, etc.

Dr. C. B. Burr said cases wherein the attacks are much longer than those the Doctor mentioned in his paper offer great difficulty in diagnosis. He had in mind the case of an inebriate where the symptoms due to withdrawal of alcohol so overshadowed those of the disease itself, a manic-depressive state, that it was difficult to arrive at a correct conclusion. However, the case was correctly appreciated in the end. He never saw

the patient in a period of excitement, but while under care the irritability and opposition, and the call for stimulants were so much in evidence that self-disparagement, hopelessness and other symptoms of the psychosis were crowded into the background. After he went away from under care, Dr. Burr received a letter from him which indicated that he was undergoing a period of excitement and since coming to this meeting, he had heard he committed suicide.

THE PSYCHOSIS OF ADOLESCENCE

By J. Montgomery Mosher, M.D.

The mental characteristics of adolescence; relations of the mental and motor mechanism of the brain to thought and action; type of abnormal action due to cerebral exhaustion; illustrative cases; prognosis; prophylaxis; results; conclusions.

Dr. Diller recalled a patient who had been under his observation many years ago, before dementia præcox had been invented, which was in many ways similar to that related by Dr. Mosher. This patient was an adolescent youth who lay in a catatonic state for a period of 18 months during which time he was entirely mute. His arms and legs placed in any position remained so indefinitely and he was apparently oblivious to his surroundings. A fly on his conjunctiva failed to produce the ordinary reflex movement. For months he was fed with a nasal tube. One morning he talked and began to move about a little. By the end of the week he was apparently entirely well. He recalled many incidents which had occurred during his illness; happily for his caretakers, he had no adverse criticisms to make of the physicians and nurses. For three or four years following his recovery Dr. Diller occasionally met this man on the street going about his business like any one else and had a little chat with him.

Dr. Mosher's discussion of the psychology of such a case is interesting and it may be adequate; but as hindsight is better than our foresight, Dr. Diller doubted very much whether armed with such knowledge we could with any degree of certainty predict recovery of similar cases. However, since his experience in this case he had never been willing to give up hope utterly for catatonic patients.

ORGANIZATION OF NEUROPATHIC AND PSYCHOPATHIC HOSPITALS WITH REFERENCE TO MEDICAL TEACHING

By Richard Dewey, M.D.

I. It is recognized that schools of medicine should give the student-body instruction in Psychoneuroses and in Psychotherapeutics.

II. Clinical instruction requires hospitals with wards similar to "Pavilion F," at Albany, or separate institutions. A rough classification will show the immense range of cases belonging to the Psychopathic Hospital: First, the psychoses in all their infinite variety. Second, the neurasthenic and hysterical. Third, the additions to drugs and alcohol. Fourth, the organic brain lesions, paralytic, paretic, arterio-sclerotic; those psychoses resulting from cardiac and renal diseases. Two great subdivisions would be the surgical and the syphilitic groups (many patients

would belong to two or more of the above groups). Fifth, the fever deliria. Finally, two important groups that psychiatry must take cognizance of, but requiring separate provision, the epileptic and the criminal, psychopaths.

Dr. Mosher said Dr. Dewey had compressed a great deal of information into a very small compass and summarized the important facts of this matter in such a concise manner we hardly realize how much ground he has covered in his digest. His own experience is principally with the annexation of a ward for mental cases to a general hospital. He had found after several years of administration they were attaining an ideal and as a general thing leveling out, so that as years go by there is less and less distinction between mental cases and cases of general disease. Patients who may come into the mental wards by reason of mental states incidental upon other conditions do better in a day or two than in the general ward. So they receive a great many cases for a day or a week or two, following an operation, and incidental upon their so called physical diseases. Of course the gratification rests in the discovery of the intimate relation between so called physical diseases and mental diseases as they have been called in the past and the more widely that idea is appreciated and the conditions are adapted to meet it the more easy and more rational will become their method of dealing with these cases. That is the point in Dr. Dewey's paper on which he touched and which Dr. Mosher thought should have emphasis.

III. The Psychopathic Hospital requires laboratory facilities, chemical, bacteriological and psychological.

IV. The question of legal commitment is here vitally concerned. Is a modification of legal procedure practicable whereby official interrogation will determine whether any individual is deprived of personal liberty, without affixing the mark of so-called insanity upon one and all?

V. Insanity is a medical misnomer.

Dr. C. B. Burr said the question of commitment is an interesting one. From motives of expediency, it is oftentimes necessary to consider more than the apparent rights of the individual. It is very comforting to a patient to know, after recovery, that there has been no adjudication of insanity and as time goes on, commitments will become fewer and fewer no doubt. On the other hand, a great deal of trouble is often escaped by early commitment.

Dr. Burr mentioned a case where a woman patient was spirited away in an automobile between two days, by her daughter, and taken to her home. Since then she has commenced proceedings for divorce, and this notwithstanding there was adjudication of insanity in Michigan. There was none in her home state and she seems to have gained the first round in legal proceedings. She has been granted alimony *pendente lite*, and apparently has full standing in court. What the future may develop, whether she will still continue to have right in court, it is of course impossible to predict, but it is obvious that had there been adjudication of insanity in her own state much trouble would have been escaped by those certainly innocent of any wrong intention in the course they adopted for the care of the patient.

PULMONARY COMPLICATIONS OF APOPLEXY.

By Philip Coombs Knapp, M.D.

Pulmonary complications add to the gravity of the prognosis in all cases of apoplexy, and are much more frequent in the fatal cases than in those which recover. Two chief types of pneumonia, the hypostatic and the inhalation forms. Neither seems to have any relation to the side of the body paralyzed, both lungs usually being affected. Prophylaxis of some importance. Danger of change of position much exaggerated.

Dr. Knapp said he had left out in his presentation of this matter the emphasis upon the mechanical production, supposing it to be obvious to the Association. Some of Dr. Mills's recommendations in regard to the conduct of the case, etc., were made by one of our honorary members many years ago in an article by Dr. Edes in *Pepper's System*. Dr. Knapp perhaps had studied Dr. Mills's book rather too hastily, but he did not think he had emphasized in his book as much as he does now the importance of frequent changes in position. Dr. Knapp thought Dr. Mills is in error when he recommends turning patients upon the paralyzed side, because the paralyzed side certainly has a little less action in very many cases than the sound side, and it increases the blood upon the paralyzed side by the respiratory muscles acting against the weight of the patient and there would naturally be less free respiration. As to the necessity of extreme care in turning, which Dr. Mills has spoken of, Dr. Knapp could not see the importance of it. Furthermore, another point which Dr. Mills has not touched upon, the importance in early stages of getting the patient in a better position to offset the action of gravity than can be gotten by the ordinary head rests, because with such head rests the body slides down toward the bottom of the bed and the patient is kept in a cramped position. Therefore, Dr. Knapp spoke of the advantage of getting them out of bed and in a sitting posture because in that way they can breathe better. He felt certain that in all stuporous cases—fracture of skull, alcoholic wet brain, apoplexy—there is an improvement in the cerebral condition by getting them actually out of bed and into a chair, and he had never seen harm come of it.

CONTRIBUTION TO THE GROUP OF HEREDITARY DISEASES:
PROGRESSIVE GLOSSOPHARYNGEAL PARALYSIS
WITH PTOSIS

By E. W. Taylor, M.D.

Report of a family in which, after the age of fifty, a slowly developing paralysis of the muscles of deglutition takes place, with coincident ocular ptosis, but without other involvement of cranial nerves. Death from starvation. So far as possible to ascertain, the disease first appeared in the mother of the patient described. In the patient's generation, two brothers, a sister, together with the patient, have been victims of the affection. Three have died, and the patient is now suffering from marked ptosis and considerable difficulty in deglutition. Another sister is still living.

Dr. Camp said he understood that, in this case, there was no atrophy and it appeared to him that the symptoms somewhat resemble those seen in the asthenic type of bulbar palsy.

Dr. Taylor said Dr. Spiller asked whether Dr. Taylor had been able to examine other members of the family. This he had not been able to do so far as this generation is concerned, since with the exception of one sister in Canada, whom he hoped later in some way to be able to learn more about, they are all dead. This sister is reported to be still well; but owing to her reticence, it is difficult to get absolute facts. Dr. Taylor was interested in what Dr. Spiller had to say regarding the question of ptosis. This in itself is not particularly remarkable, but in combination with the glossopharyngeal paralysis constitutes a grouping which is apparently unique. None of the other cranial nerves in the case which he had carefully examined are in any way involved. The knee-jerks are perfectly normal, as are the other reflexes of the lower extremities. There is, therefore, no evidence whatever of involvement of the pyramidal tracts. There had been no other diseases of a motor sort in the family so far as ascertained. With regard to Dr. Camp's suggestion, that possibly the condition is one of myasthenia gravis, it may be said that this is altogether improbable because of the unique and constant localization of the paralysis in many members of the family, because the affection is steadily progressive, and because in no other regard does it in any way resemble myasthenia. Furthermore, the fact that it occurs only after the fiftieth year is strongly against the hypothesis of a myasthenia, as is also its manifestly hereditary or family character. Even were it to be included in this group, it would be none the less remarkable on that account. The condition is presumably a degenerative one, doubtless involving the nuclei of certain cranial nerves, analogous perhaps to progressive bulbar palsy, but differing in the nerve involvement, which so far as Dr. Taylor was aware has not hitherto been described.

THE PATHOLOGY OF TABETIC OCULAR PALSIES

By William G. Spiller, M.D.

Difference of opinion exists as to the nuclear or nerve origin of tabetic ocular palsies, and a distinction is made in the pathology of the ocular palsies of tabes and brain syphilis. The author has studied with microscopical serial sections a case of complete ophthalmoplegia in tabes. The subject brings into consideration the relation of parasymphilis to syphilis.

Dr. Fry asked whether in these third nerve palsy cases there is not more pain in the "syphilitic" ones than in those we have been in the habit of calling "post-syphilitic" from a clinical standpoint.

Dr. Fisher asked Dr. Spiller, as in many cases of tabes we have these early ocular palsies which just as in syphilis disappear and often never return, whether that fact does not indicate that the lesion affects the nerve roots rather than the nuclei.

Dr. Spiller thought that the so-called parasymphilitic affections of the brain are of more chronic type and therefore less likely to be with pain but many of the "syphilitic" ocular palsies are painless.

He believed a complete recovery would be more probable from a nerve lesion, but of course recovery is possible from nuclear lesions if they are not too severe, as is seen in poliomyelitis.

MUSCLE TONICITY, EMOTIONAL EXPRESSION AND THE CEREBRAL TONECTIC APPARATUS

By Charles K. Mills, M.D.

Dr. Mills first said that since he had sent the announcement of his paper to the secretary some month or two before the meeting he had changed the title in the course of the development of the article. At first this title referred primarily to one of the most dramatic symptoms exhibited by the patient—involuntary painful emotional expression—but his further studies of the case and of the literature of the subject had caused him to expand the scope of his paper for presentation and publication. One of the fundamental ideas in this paper was that of the existence of a special cerebral tonectic apparatus. This on its motor side is extra-pyramidal. He held in his paper that tone derived from sensation and idea has in the cerebral cortex a special rendezvous. He believed that this was largely midfrontal and prefrontal and more highly developed in the right cerebral hemisphere.

Motor centers of the cortex cephalad of the central fissure had cellular tonectic annexes which together composed a tonectic or tonecto-motor zone. By association tracts this tonectic area was connected with the striatum so that its impulses here grouped could be delivered from the strio-thalamic system en masse by way of the cerebello-rubrothalamo-cortical system on the pyramidal motor apparatus. In this way the tone necessary to the steadying of synergic motor activity was acquired for normal movement. When this rhythmic tonectic influence was withdrawn—by large lesions of both lenticular nuclei for instance—the cortical tonectic centers released from their striate connections discharged directly by gyral associating tracts on the cortical motor centers and thus arose the phenomena of hypertonicity, or perhaps it might be better to say of irregular tonicities.

Dr. Mills believed that the caudatum was more especially concerned with the tonectic phenomena of the autonomic or sympathetic nervous system and that its influence was exerted by way of the nucleus ruber and the cerebello-rubro-spinal tract.

The paper contained numerous observations on a case of bilateral caudato-lenticular degeneration or destruction with a microscopic study by Dr. Spiller of the secondary degenerations resulting. In the first place, the patient exhibited to a very large extent the symptom-complex of Kinnier Wilson's bilateral lenticular degeneration—involuntary emotional expression, hypertonicity, tremor, involuntary movements, contracture attitudes, dysarthria, dysphagia, some mental reduction and, late in the case, rigidity. Sensory symptoms were entirely absent. While the deep and superficial reflexes were very prompt, the Babinski extensor phenomenon was not present on either side.

In addition to this lenticular syndrome the patient exhibited vasomotor phenomena especially showing themselves in a congested appearance of the face. The temperature ran for months a degree or so below the normal, and respiration and pulse were in excess of normal rates. There was some irregular or peculiar impairment of control of the bladder and bowels.

Besides the symptoms just enumerated the patient had a few others of pyramidal type, the necropsy and pathological examinations showing limited involvement of the posterior limb of the internal capsule.

Dr. Mills discussed to some extent the literature of the subject of muscle tonicity and emotional expression and called attention to the records of some cases of large lesions of the midfrontal and prefrontal areas of one or both hemispheres in which phenomena somewhat similar to those produced by large striate lesions were present.

He held that tone as exhibited in man was a cerebral cortical function and that the three great elements represented in normal and abnormal movement were energy derived through the pyramidal apparatus, synergy from the cerebellum, and tone through the cerebral tonectic apparatus.

Dr. E. M. Williams said he had recorded a case of paresis, in his paper at this meeting, in which there was an incomplete left hemiplegia clearing up in the course of the day, the right side then becoming affected and clearing up with a later involvement of the left side. In addition there were generalized myoclonic twitchings, on the third day beginning to affect the face so that the facial muscles on the left jerked two or three times per second. During periods of about a half to two minutes what seemed to be outbursts of laughter occurred and during these the patient made peculiar grunting sounds.

The patient died and Dr. Williams could not get a necropsy. He thought it interesting as possibly further affirming the James-Lange theory of the dependence of emotion upon the motor stimulation. Whether the emotional outbreaks were primary in this case or secondary to the facial movements could not be decided.

THE IMPORTANCE OF THE BONY SINUSES ACCESSORY TO THE NOSE IN THE EXPLANATION OF PAIN IN THE HEAD, FACE AND NECK

By M. A. Bliss, M.D.

Not infrequently are pains in the face, head and neck treated for long periods with various internal remedies when the explanation lies in the accessory sinuses, and the ganglia and nerve trunks adjacent thereto.

A rather clear symptom complex springs from the district of the sphenopalatine ganglion, and alcohol injected therein cures it.

Incessant nuchal pain springs from the post ethmoidal sphenoidal district. Pronounced headache, dizziness, nausea and optic swelling arise also from this field. Cases illustrating these statements will be cited.

The ordinary casual examination of the nasal district does not suffice in this work.

The rhinologist must make numerous and painstaking examinations before the field under consideration may be excluded.

Dr. Hecht said this paper calls attention to manifestations which are too frequently accepted by physicians and neurologists as well, as expressions of a neurosis. This point of view, he thought, arises from the fact that the chief complaint—that of pain—is so long protracted, troublesome and resistant to treatment. In cases, for instance, of supraorbital neuralgia associated with frontal sinus infection, where all traces of pus have disappeared, the mucosa continues to present a chronic inflammatory appearance and the neuralgia continues in the presence of this slight evidence of pathology. In short, the presence of pus is not necessary to the inference of cause and effect. Neuralgias are readily superimposed upon pyogenic infections long after all traces of pus have disappeared.

In addition to the neuralgias, Dr. Hecht called attention to the myalgias that so frequently follow in the wake of infections and sometimes locate at points quite remote from the original source of infection. He had observed several instances of nuchal muscle pains and occipital neuralgias which he felt could be definitely traced to infections of tonsil crypts and pyorrheal pockets.

Arthritic manifestations following these sinus infections are not at all uncommon.

After all, it is a matter of a low grade of sepsis, with shifting areas, at times sharply circumscribed, of pain or tenderness of muscles, nerves and joints.

In the course of their chronicity, he had known patients to get much depressed as the result of the nagging, distressing, degree of the pain resulting from these sinus infections. At times, these supraorbital neuralgias behave much like the attacks of migraine that have their onset in and above the orbit, and particularly in one young woman whom he saw very recently, and who was known to have a migraine for years, it was difficult to differentiate the supraorbital type of neuralgia which followed a long standing frontal sinus infection.

Dr. Hecht asked Dr. Bliss what use he had made of autogenous vaccines for these complications, and, if any, with what result.

Dr. Fry said the study of some of these cases is difficult indeed, requiring great patience on the part of the rhinologist. Everyone of them should be watched by a neurologist, as well. Dr. Fry was familiar with the work Drs. Sluder and Bliss had been doing and with some of their cases. It seems there is much promise in it.

Dr. Bliss said in response to Dr. Hecht's question they had not used autogenous vaccines.

RECURRENT MENINGITIS, DUE TO LEAD, IN A CHILD OF FIVE

By H. M. Thomas, M.D., and K. D. Blackfan, M.D.

Boy, five years old, admitted August 22, 1913; comatose, slight fever, headache, convulsions, vomiting, rigidity of neck, strabismus, unilateral choked disc with hemorrhagic retinitis, etc. Von Pirquet negative; leucocytosis 27,600; Wassermann in blood negative. Lumbar punctures. Clear, sterile fluid, 20 to 40 cells; Noguchi positive; Fehling solution reduced; Wassermann negative.

Improvement rapid; in 11 days seemed nearly well. Discharged September 20, in good condition. Eyegrounds normal. Child kept under observation. Condition remained good for five months. January, 1914, severe nose bleeds.

Re-admitted to wards, March 3, 1914, after three days of headache, vomiting, stiff neck, and 12 hours of coma and convulsions. Condition quite similar to that on first admission. Wassermann in fluid positive with other antigen.

Typical lead line discovered.

R. B. C., 4,480,000; W. B. C., 23,100; hemoglobin, 55 per cent. Marked stippling of white cells. (Grawitz granules.)

Source of lead discovered in child's habit of eating paint from bedstead. Discussion of case. Lead probably a more frequent cause of intra-

cranial disease than is usually recognized. Interest of the French in meningitis due to lead. Hardly noted in German, English and American literature.

Dr. Bliss said they had a large lead district near St. Louis and this paper reminds him of the fact that in the manufacture of lead, and products of lead, they had always an extraordinary number of cases of epilepsy. Dr. Thomas did not mention in his paper whether this child under his care had repeated convulsions, but they had been accustomed to look for lead poisoning in epileptics. Especially in those who work in the lead industry.

Dr. Allen called Dr. Thomas's attention to a report by Dr. Spiller on meningeal involvement due to lead poisoning.

Dr. Thomas said he knew of Dr. Spiller's case of lead encephalopathy in which were found, among other lesions, small cell infiltration of the meninges. This interesting case and other similar cases have been collected in a German thesis.

The cases referred to by Dr. Bliss are most interesting, and it would be important if in them the lumbar puncture revealed irritation of the meninges.

Dr. Edward M. Williams read a paper with the title: "Paramyoclonus Multiplex, Including a Case With Necropsy Showing Lymphatic Infiltration of the Pia." (See this *Journal*, 1914.)

Dr. Starr said he would hesitate, of course, to throw any doubt on the diagnosis of the case reported, but it differed so essentially in all its particulars from the well-marked group of cases of paramyoclonus that it seemed to him a little questionable whether it should be assigned to this class. The peculiarity of the spasm in paramyoclonus is that it is confined to muscles connected with the trunk, there being no spasm in muscles not connected with the trunk, such as hands or toes. Very frequently in paramyoclonus there is a history of functional nervous disturbances which in the course of time gradually pass off. Dr. Starr did not think there is any case on record where a disturbance of speech has been a marked symptom. He did not think in the literature on the subject with which he was thoroughly familiar, there had been any record of epilepsy in paramyoclonus. He had seen quite a number of these cases and they did not correspond to the case described by Dr. Williams.

Dr. Williams said he thought the case was rather doubtful. That was one of the reasons he reported it as he could not classify it definitely as one thing or the other, but it seemed to be nearer paramyoclonus than anything he could think of.

He realized the fact that the movements of paramyoclonus multiplex are supposed to be confined to the trunkal muscles. He believed, however, that the arms were involved in the earlier reported cases.

What he meant to bring out particularly was the fact that these cases either are hysterical or symptomatic of some other disorder, and he could not see why they should be classed as definite clinical entities.

The case in his paper referred to by Dr. Hun was not a purely hysterical case, but one of unmistakable epilepsy, with an additional hysterical element.

UNILATERAL HYPERTROPHY OF LIMBS. REPORT
OF A CASE

By Howell T. Pershing, M.D.

Girl, aged 20. Mother had Raynaud's disease. Greater size of right limbs first noticed at 3 years. Difference has gradually increased, most rapidly at puberty. Mental development normal. Increased size principally in thickness of subcutaneous tissue. Measurements, photographs and radiographs. Theories of pathogenesis.

Dr. Allen called Dr. Pershing's, as well as Dr. Moyer's attention to the fact that not only does this process involve the soft tissues but that by his measurement of the X-ray picture with an accurate metal rule Dr. Allen found quite a difference in the diameter of the tibia. The difference in diameter amounting in one place to 4 mm. and in another place to 7 mm. This means a discrepancy in circumference too great to be left out of consideration.

Dr. Peter Bassoe, Chicago, stated that he regretted that Dr. Pershing uses the term unilateral hypertrophy of limbs rather than trophedema. In the small but well-defined group of cases hitherto reported as unilateral hypertrophy, or hemihypertrophy, there has been a distinct hypertrophy of all the tissue elements on the affected side. Thus in the case reported two years ago by Dr. Bassoe, not only were the extremities longer on the affected side, but the circumference of the chest was greater, the teeth and gums were larger and the nostrils were larger on the hypertrophic side. In the reported necropsies, the last case being that of Cagiati, the paired organs have also been found larger on the hypertrophic side. It would, therefore, appear preferable for the case just reported by Dr. Pershing to be grouped under the heading of the trophedema of Meige.

Dr. Archambault said there was a certain discrepancy between the bony development on the two sides. In the photographs of the chest there was a very decided difference between the sternal portions of the clavicles and the resulting prominence of the sterno-clavicular joint on the affected side is most striking.

Translations

VAGOTONIA

A CLINICAL STUDY

BY PRIVATDOZENT DR. HANS EPPINGER AND DR. LEO HESS

OF VIENNA

TRANSLATED BY WALTER MAX KRAUS, A.B., M.D., AND
SMITH ELY JELLIFFE, M.D., PH.D.

(Continued from vol. 41, page 998)

It is remarkable that we often find some evidence of a persistence thymus. Many vagotonics are of lymphatic constitution, as shown by the enlarged tonsils, large lingual follicles, as well as the solitary lymph nodes of the tongue, and by the condition of the lymphatic apparatus of the conjunctiva. Occasionally rectoscopic examination will reveal large lymphatic follicles on the rectal mucous membrane. As has been stated the tonsils are large, pale pink and much pitted.

We know full well that associated with the lymphatic constitution and persistent thymus is a diminution of the resistance to infection. The vagotonic also has a tendency to colds and other infections, and when infected has but very little resistance. Later on we shall discuss the possible etiological relationship between thymus persistence, the lymphatic constitution, and the vagotonic constitution. We may say at this point, however, that on X-Ray examination we have been able to find a narrow aorta in a group of our vagotonic cases. We not only found anemia in this group of cases but also in vagotonics in general. The criterion for this was not alone the facial color, but the additional evidence of a blood examination. In fact a marked grade of anemia is quite characteristic of the vagotonic constitution.

When we discover that vagotonia is associated with so many other constitutional anomalies, which are manifestations of inferiority, we naturally are inclined to ask whether vagotonia itself is

not a form of constitutional inferiority. That this view is true is strengthened by the fact that vagotonia is so frequently found in classes of people who show signs of degeneration, as, for example, Polish Jews.

Our contention that many diseases are related to the vagotonic constitution and that certain signs and symptoms are only produced when the proper soil is present admits of certain exceptions. As examples of this are certain cases of typical general vagotonia, associated with hyperacidity, diminished peristalsis and diminished gastric tone. It is also well known that spastic constipation, which is so frequent a finding in connection with these signs of autonomic stimulation, may be associated with gastric atony. There are some cases in this group in which the gastric secretion is diminished. Finally tachycardia is often found present, while the gastro-intestinal tract and other visceral organs show signs of autonomic hyperactivity. These facts raise the question, can vagotonia exist in some parts of the visceral nervous system, while the reverse exists in others? Before entering into a discussion of this matter, we must outline our definition of vagotonia very sharply, and we must state that if we should find signs or symptoms which are significant of autonomic stimulation, as, for example, gastric hyperacidity, or nervous diarrhea, we would not feel justified in making vagotonia responsible for this symptom, without further inquiry into the case. For this symptom may be due to some organic condition, just as bradycardia may be of organic origin. Furthermore the symptoms referable to various organs, which are signs of autonomic stimulation, must have been spontaneously produced and not have been brought out by the action of drugs.

Thus we may say that vagotonia is a purely functional disease, not referable to any organic basis, which does not affect one organ alone, but spreads out, even if but transiently, in several branches of the autonomic system, until finally it involves them all and affects the entire autonomic system.

This definition of the condition as a functional disease narrows down the number of diseases which when having signs of autonomic stimulation might be considered as vagotonic. Looking at the matter in this way we are not inclined to consider every spastic state of the autonomic nervous system as a part of general vagotonia, even though it may be referable to autonomic stimulation,

for if we did this and went so far as to say that every spastic state, which was in any way related to the vagus, was a sign of vagotonia, we should have to divide all people into the vagotonic and sympathicotonic, a procedure which would lead us too far afield. On the other hand if we did try to enlarge the narrow limits we have set for vagotonia and added to the not infrequent cases of this condition those which showed some signs which through being opposite to what was expected, spoiled the picture, it would but serve to increase the interest in this most important constitutional anomaly and throw more light upon visceral neurology.

Since true vagotonia may be associated with diseases of other etiology, organic or functional, certain groups of symptoms may appear in a given case, some of whose individual components will not fit into the picture of generalized vagotonia. Enteroptosis is very often associated with vagotonia and may be the cause of symptoms quite different from it. The tone of the organs within the abdomen, as, for example, the stomach, receives considerable aid through that of the abdominal wall. Should this latter be absent the smooth muscle cells are robbed of a valuable support. Vagotonics are just the class who, in connection with the symptoms of autonomic hyperactivity, suffer from various other complaints and pains, a fact which readily leads the patient into a state of malnutrition. The malnutrition affects the entire body, and this may affect the smooth muscle cells of the stomach. In a group of cases we saw that increase of the state of nutrition and general constitutional improvement bettered not only the gastric tone but also the secretory activity of the stomach.

Further difficulty in making a clear cut diagnosis of vagotonia is found in the symptom, tachycardia. This, we believe, is present in some cases due to enteroptosis, a relationship, which is most striking in cases of "*cor pendulum*." These people, who in every other way are true vagotonics, usually have a normal pulse rate when they are recumbent and breathing normally. However, the slightest exertion,—a tachypnoe of nervous origin for example—may cause a transient dyspnea which is augmented by the increased tone and relative narrowness of the bronchial branches. In enteroptotic individuals with long thoraces, this is followed by a very low position of the diaphragm, which in time, separates the heart from its proper support. This low position of the dia-

phragm, as well as changes in the position of the heart itself, are surely sufficient cause of a compensatory tachycardia. If one considers furthermore that secondary anemia is frequently present, and that autonomic stimuli may appreciably alter the distribution of the blood, it is scarcely to be wondered that this most important organ should exert itself in the interest of increased vitality. Only when the proper physiological requirements for the circulation are present, does the heart respond to autonomic stimulation.

7. LOCAL VAGOTONIA

In describing the syndrome of general vagotonia, we observed that an increase of tone could also occur in isolated branches of the autonomic system. This observation may not be in accord with what we have said up to now about localized vagotonia, but the fact remains that in the absence of local causes for the condition, a general state must be assumed of such a nature as to cause an increased tone in but one part of the autonomic system. Furthermore this state may exist even if there be no observable signs of disease or of increased irritation. Pharmacology has taught us that the various vagotropic substances have varying affinities, i. e., they affect different parts of the autonomic system with varying intensity [atropin for example]. So also, we believe, do metabolic poisons act, some causing an increase in tone in this part of the autonomic, some in that. It is in this sense only that we have postulated a local vagotonia.

It is to us a matter of no small interest, that on the basis of clinical observations, we have been able to find spastic states, affecting isolated parts of the autonomic, which may justly be called of true autonomic origin, and which seem to be associated with similar states in the particular branch affected.

(To be continued)

Periscope

Brain

(Vol. 36, Part 2)

1. Experimental and Pathologico-Anatomical Researches on the Corpus Callosum. C. T. VAN VALKENBURG.
2. Sensory Changes in Friedreich's Disease. P. W. SAUNDERS.
3. The Wassermann Reaction and Its Application to Neurology. PAUL FILDES and JAMES MCINTOSH.
4. On the Bulbar Nuclei, with Special Reference to the Existence of a Salivary Center in Man. ANTHONY FEILING.

1. *Researches on the Corpus Callosum.*—On separating the two fore-brain hemispheres of any mammal, the corpus callosum becomes visible; its position between the two halves of the brain at once suggests that it serves as a connecting link between the hemispheres. There is no doubt that certain bundles exist in this mass of fibers, the origin and termination of which lie on the same side of the middle line. These are the fibers which belong, in the wide sense of the term, to the olfactory system, i. e., the fornix longus, tænia tecta, and the nervi Lancisii. These, however, the author says, do not come within the province of his research, which is confined to what is commonly understood as the corpus callosum, that is to say, the chief hemispherical commissure of the neopallia. The term commissure may be understood to mean a connection of corresponding regions of the opposite hemispheres; or, taking it in a broader sense, a connection in general of the one half of the brain with the other, through which both homotopical and heterotopical cortical regions are associated with each other. To give the word its full meaning it connotes an interhemispherical association system. Both interpretations are found in the literature, though it is not possible to state that they always rest upon conclusive anatomical or pathological evidence. The more modern writers embrace the latter opinion on the whole, chiefly on the strength of Cajal's histological investigations; in small mammals, as the mouse, with the aid of his silver methods, Cajal found that cortical axones arise, which either directly or as collaterals of projection and longer association neurons, pass to homo- and heterotopical points of the opposite hemisphere through the corpus callosum.

The author, however, states his conclusion that the ending of the callosal fibers cannot be positively proved from normal silver preparations. Moreover, were normal histological investigations sufficient to place Cajal's theory beyond all question, they afford very little evidence of the "associative" nature of the commissure. Cajal believes that the callosal fibers give off collaterals before ending, but these can be followed with certainty over very insignificant distances only in one section. Though strictly speaking the term association may be applicable to such a connection, it approaches more nearly to what one may consider as a commissure. In any case it is evident that Cajal's theories, although they may be perfectly correct, by no means justify the frequent assumption that heterotopical

cortex regions of both hemispheres stand in connection by callosal fibers with each other.

The author believes he is justified in stating that in the posterior central convolution (1) The relation of the origin and termination of the callosal fibers is not definitely local. (2) The ending of these fibers is relatively diffuse. (3) Fibers end here which come from the contra-lateral gyrus centralis anterior, and to some extent at least the origin and termination both in front and behind the sulcus centralis are situated in the same horizontal level.

On the other hand, the anterior central convolutions are:

(1) Connected homotopically, in the sense that the origin of corticofugal fibers is more or less surrounded by a radiation of fibers from the homologous spot on the opposite side; it is doubtful if there exists a heterotopical connection between the gyri centrales anteriores; the author has no argument in favor of it.

(2) They are connected with the contra-lateral gyrus centralis posterior. The fibers which form this heterotopical connection are apparently far more numerous.

Commenting on clinical pathology, the author says that it is remarkable that of all the symptoms described in cases of callosal lesions very few may be ascribed, with any degree of certainty, to destruction of the commissural connections. Perfectly isolated callosal lesions rarely, if ever, occur, and the total absence of this great commissure does not necessarily cause any typical symptom. The symptomatology is very variable. Practically only those cases come under consideration where either an isolated, or more or less local, callosal softening existed, or in which by a softening in one hemisphere, a function is disturbed, which is not under the control of that hemisphere alone. Such disturbance, when remote influence by a tumor, etc., may be excluded, can only be explained through the commissural fibers.

The author believes that in the first place his investigation offers an anatomical explanation of the well-known fact that in cases of cortical epilepsy the excitation of a motor center in the affected hemisphere can cross through the corpus callosum to the homologous center in the healthy hemisphere. If the progression of the convulsions from the one side to the other is not accurately homotopical, as is indeed often the case, it may be assumed that the excitation of the center first affected was so strong that the irritation spread directly from here to the homologous region of the opposite hemisphere.

Indirect apraxia (*apraxia callosa*) of the left extremities which Liepmann has described, is assumed to be due to a lesion of the commissural connection between the left and right sensori-motor centers. This view, put forward by Liepmann, is closely connected with his statement that the left sensori-motor centers have a primary rôle in evoking more complicated actions in general; even the movements necessary for given action of the left limb are innervated from the right hemisphere. The result, the author says, of his anatomical investigations, that there exists a connection between the gyrus centralis anterior and the two contralateral central convolutions, may assist in the explanation of the fact that this direct apraxia itself can be considered as a "cortical" disturbance ("*gliedkinetische, innervatorische Apraxie*"). The movements of the left arm then are not disturbed in consequence of a deficient formulation of the "psychic plan," nor by the ineffective transmission of the "plan" to the region, whence the execution takes place finally, but by the inadequate execution of the

action itself, as this depends on the adequacy of the immediately preceding intra-cortical activity; but this must not be confused with the regulating impulses from the immediate proximity, the disturbance of which produces cortical ataxia. In the reported case in which only two handed movements were disturbed, it was because not only did these need the coöperation of the apraxic right hand, but the functions of the left hand were primarily affected. All remaining actions of the left hand, as far as they could be examined in the aphasic patient, were quite normal.

In addition to the complete apraxia, there was a strong disinclination to make any movement of the right hand or arm. Whether this innervation was affected by the extensive centro-parietal, or by the frontal softening, as Förster and others seem to believe possible, it is certain that the initiative of the left arm and hand was not lessened by these large lesions. There can be, in any case, no question of a unilateral "eupractic center."

Finally, there is the question as to the causes of mind blindness. A lesion of the posterior part of the corpus callosum is generally considered, at least partly, responsible for this. If this assumes that it is due to the commissural connection between two primary visual fields (*area striata*) being disturbed, the view is untenable, as it is an anatomical fact that no such connection exists. If the chief cause of mind blindness is to be sought in a lesion of the callosal fibers, it must be of those that connect other parts of the two occipital lobes, and it is certain that fields 18 and 19 (Brodmann) receive and give rise to parastriate radiations. These regions of the cortex comprise the "visuo-psychic area" of the English authors. It is certain that these fields coöperate with the *area striata* in the higher visual functions. They are, moreover, phylogenetically more recent than field 17, and increase relatively in the higher mammals. Parallel with this the occipital callosal fibers become more abundant. The exactness of the hypothesis of Niessl v. Mayendorf, that in visual function the left hemisphere predominates over the right, and localized softenings of the pole and ventral part of the occipital lobe ("left macula center") may cause mind blindness, can be determined only by fuller clinical and anatomical investigations. One will then have to reckon with the fact that the *area striata* has no commissural connections.

2. *Sensory Changes in Friedreich's Disease.*—The sensory changes, says this author, have been studied by several observers. Noica, in two cases of the disease that he examined carefully, found very considerable loss to different forms of sensibility. Egger, in his investigations of "osseous sensibility," considered that this is regularly affected in Friedreich's disease. Other investigators have from time to time reported cases in which a certain amount of sensory loss was present, and cases indeed with some sensory loss have been mentioned incidentally from the time of the early differentiation of the disease from *tabes dorsalis*, and its occurrence is referred to in most neurological text-books.

The classical description of the disease, however, while admitting that loss to some or all forms of sensations may occur, regards it as altogether an unusual and exceptional feature, notwithstanding the fact that one of the great sensory plants of the cord is the chief site of the pathological changes that are present in the disease. A series of systematic observations, therefore, on a considerable number of cases of Friedreich's disease would seem justified, in order to see how far the classical description of the disease holds in the light of our present knowledge concerning the different forms of sensibility.

The author's chief aim in undertaking his work was the important

question of sensory conduction in the cord, as the more recent work of Petré, Rothmann, Head and his collaborators, and other investigators make it probable that impulses underlying the sense of position and appreciation of passive movement, the recognition of two compass points simultaneously applied to the surface of the body, the appreciation of vibration, and the recognition of size, shape, form and weight, and possibly of roughness and texture, are conveyed through the dorsal columns, tracts of which there is systematic degeneration in Friedreich's disease.

Most investigators now agree with the view that the dorsal columns furnish the principal pathway in the cord for the conduction of impulses that subserve muscle sense, meaning by that term chiefly the sense of position in space and the recognition of passive movement. Petré concludes from his examination of cases of stab wounds involving the spinal cord that these impulses pass up the cord uncrossed in the dorsal columns and in the dorsal spino-cerebellar tracts, but the view of Head and Thompson, based on their investigation of the grouping of sensory elements in Brown-Séquard lesions, that the dorsal columns subserve the conduction of the impulses underlying the recognition of position and movement that pass to consciousness, while the spino-cerebellar tract is concerned with those impulses that are responsible for the unconscious maintenance of equilibrium, seems likely to be the more correct, and Thompson has adduced later clinical and pathological evidence from a case of combined degeneration to show that the sense of position and recognition of passive movement may be grossly affected by a lesion limited to the long fibers of the dorsal columns.

Head and Thompson have shown also that the recognition of compass points simultaneously applied is dissociated in the cord from tactile sensibility, and passes up uncrossed in the dorsal columns, while Bing has brought evidences that the impulses that subserve the appreciation of vibration also pass up the homolateral side of the cord, and therefore must be conducted by the dorsal columns.

The investigations of Head and Holmes on Brown-Séquard lesions, cited in their paper go to show that not only do the knowledge of position and passive movement, the recognition of two points, and the appreciation of vibration depend on the integrity of the dorsal columns, but that the recognition of size, shape, form and weight, and the appreciation of consistence and texture are affected in common with these other elements of sensibility, and the impulses which underlie them would seem, therefore, also to be conducted by the dorsal tracts. Thompson also, in his case, noted that the recognition of weight was grossly affected, as well as the appreciation of position, movement and double contacts. Moreover, the author speaks of an unpublished case of cervical Brown-Séquard lesion, studied by Head and Holmes, which goes to confirm the view that the impulses which underlie recognition of size, shape, form, weight, consistence and texture, as well as the appreciation of position, movement, simultaneous contacts and vibration, are conducted through the homolateral side of the cord.

The impulses of light touch and pressure touch are, according to most of these observers, associated together in the cord. These impulses are conducted partly by an uncrossed path in the dorsal columns, mostly by a crossed path in the lateral columns (Petré), or in the ventral columns (Rothmann). The localization of tactile sensibility is associated with touch and is mainly the crossed pathway. All impulses of whatever kind that are painful, all heat impulses and all cold, cross almost at once after

a relay in the dorsal horns, and pass up the cord in the opposite lateral column, though possibly some of them pass without crossing by the lateral column of the same side. Systemic degenerations of the dorsal columns are not numerous. *Tabes dorsalis*, it is generally recognized, is, at least predominantly, a disease of the dorsal roots. In combined degeneration the process is probably not a true system degeneration but a funicular myelitis (Nonne). In *Friedreich's disease*, however, we have a condition which is usually regarded as essentially a systemic degeneration of the dorsal columns, even although the pathological changes are not limited to them. The changes in the dorsal column tracts are the most constant and most pronounced features of the pathological anatomy of the disease, and are present in all cases. In the sacral and lumbar regions these tracts may be almost entirely degenerated, but higher in the cord the degeneration in the column as a whole is not so marked. In the dorsal and cervical regions, the root-entry zone seems considerably less affected than the rest of the column; and the cornu-commissural zone, the areas immediately next the grey matter of the dorsal horns, and *Lissauer's tract* may be scarcely at all affected. The degeneration in the other parts of the column, however, remains very severe throughout the whole cord. The long and medium fibers of the dorsal columns are therefore the ones chiefly affected, and the affection of these may be so intense that scarcely a fiber may remain visible in cross section.

In the other tracts involved in the disease, the pyramidal and the spino-cerebellar, the degenerative changes are rarely so great as in the dorsal columns. The affection of the pyramidal tracts is most intense in the sacral and lumbar regions and diminishes as we pass up the cord, though in some cases it may be traced as far as the pons or the lower part of the crus. The degeneration in the spino-cerebellar tracts, on the other hand, increases in extent and degree, from below upwards and becomes most pronounced in the higher cervical segments.

As contrasted with the great affection of the fibers in these different tracts, and especially in the dorsal columns, the changes in the cells are not so frequent or so pronounced except, perhaps, in *Clarke's column*. In some cases the spinal ganglion-cells are slightly atrophied and the dorsal spinal roots, and even the peripheral nerves may be involved. There is, however, no reason to consider that the affection of the dorsal columns is secondary to a primary degeneration of the ganglion-cells or of the roots, for the changes in the roots are not nearly so definite, so frequent, or so intense as in the dorsal columns, and the affection of the ganglion cells is slight and inconstant.

The widespread nature of the pathological changes in this disease and its variable incidence on different systems of fibers in the cord are in harmony with the view that it is a true systemic degeneration of the long spinal tracts, and especially of the dorsal column system. But even if the degeneration in the dorsal columns be regarded, not as a primary systemic one, but as due to an elective or chance degeneration in the dorsal roots, the existence of profound changes in these columns is by far the most outstanding feature of the pathology, and we should expect, therefore, to find in *Friedreich's disease* those forms of sensibility disturbed which have been assigned to the dorsal columns, and disturbed in proportion to the intensity of the dorsal column affection.

For the purpose of investigating this, the author examined twenty cases of *Friedreich's disease*, and found that all the patients examined presented the usual signs of *Friedreich's disease*, the skeletal deformities, the

absent deep reflexes, the extensor plantar responses, the nystagmus and the ataxia so characteristic of the disease.

The methods which he used were mainly those employed by Head and Holmes in their work on sensory changes in cerebral lesions in as far as these were applicable. They had to be modified somewhat, because the symptoms in Friedreich's disease being bilateral, there was no sound side which could be used as a standard of comparison to estimate the amount of loss present as in Head and Holmes's work, where the disturbance was unilateral.

He concludes that, in this disorder, the appreciation of touch, pain and temperature are very irregularly affected in the upper extremities, never more than very slightly and often not at all. When loss occurs it is almost always a slight distal blunting to touch, and very rarely to pin prick, or to heat and cold as well. In the lower extremities these cutaneous elements are more frequently involved, and there is often some distal hypoesthesia to light touch or even to pressure touch, and in many cases also to pin prick and to heat and cold. It is the other elements of sensation, however, chiefly those that are commonly included in the term "deep sensibility," but also the appreciation of simultaneous contacts, and of size, shape, and form, that are most severely affected. The sense of position and the recognition of passive movement, the appreciation of double contacts and of vibration are almost always more or less affected, especially in the distal parts of the lower limbs, and not infrequently, though to a less degree, in the upper limbs as well. The frequency and the constancy with which these three elements of sensation are disturbed are indeed characteristic of the disease.

The recognition of shape and form, and the appreciation of size and weight are also affected, but much less frequently and less seriously than the sense of position, the appreciation of compass points, or of vibration. This is probably due to the fact that these forms of sensibility can be examined in the upper extremities only where the sensory loss as a whole in the early stages of the disease is usually not great, and possibly also to the fact that they are more elaborate forms of sensibility. They would seem, however, to be associated in their disturbance rather with the loss of the sense of position, the appreciation of double contacts and of vibration than with tactile loss. The recognition of shape, and that of size and of weight are affected with the same frequency, and usually in the same patients, while the recognition of form is less often disturbed.

The elements of sensibility, therefore, which it is assumed are conducted through the dorsal columns of the cord, would seem to be disturbed in Friedreich's disease more or less constantly and characteristically. The loss, especially in the early stages of the disease, falls most heavily on the lower extremities, that is on the regions innervated by the spinal segments that are earliest affected, and tends to spread higher and higher over the body and to become more profound as the disease advances.

Those elements of sensibility that are conducted through the dorsal columns have been divided into two groups. The recognition of two points of the compass, and the appreciation of size, shape and form which are dependent at the periphery on the integrity of the cutaneous nerve-endings would seem to run in one group, and the sense of position and of movement, the recognition of vibration and the appreciation of weight, which are dependent peripherally on nerve endings in joints, tendons and muscles, apparently compose another group. As the disturbance of the sense of position, of double contacts and of vibration was always so great

in every case in which the recognition of size, shape, form and weight was affected he found it impossible to correlate the disturbance in recognition of size, shape, and form with that in the recognition of compass points particularly; of the loss in the appreciation of weight with that of position. In the description of the fate of these elements of peripheral sensibility, therefore, he preferred for convenience to follow the order in which they were tested clinically, and to consider together the disturbance of the sense of position and of movement, the loss of appreciation of double contacts and the diminished recognition of vibration which constitute a more primary group; the recognition of size, form, shape and weight, would seem to compose a group of most secondary and elaborate sensations.

In a disease such as Friedreich's ataxia where there is a very slow progressive degeneration of the dorsal column of the cord, it is inevitable that though the sensory loss may conform to a definite type there must be much variability in the relative intensity of the affection of the different elements of sensibility in different cases. The frequency, however, with which a characteristic sensory loss does occur would seem to justify its inclusion in the clinical picture of the disease as an integral, not as an exceptional, feature; and further, the constancy and regularity with which this loss involves the sensory impulses assigned to the dorsal columns of the cord goes to confirm the conclusions regarding sensory conduction in the cord that have been obtained by the clinical and anatomical study of local spinal lesions.

3. *Wassermann Reaction in Neurology.*—The authors' intentions in writing this paper were to review the present state of knowledge of the Wassermann reaction in all particulars which bear directly or indirectly upon neurology, but in these particulars only. They have also described fully the methods utilized by them as being the most satisfactory according to their deductions. These deductions are based upon researches into the subject of syphilis generally, and also upon the results of a long collaboration with Head and Fearnside upon syphilis of the nervous system in particular.

The work is treated largely from a technical point of view, and thus assumes a general knowledge of the principles, and even of the technique, of the reaction on the part of the reader. The theory of the subject is not discussed, since that has no application to neurology, neither have the modified tests been reviewed.

The paper is intended to be read in conjunction with works published or forthcoming by Drs. Head and Fearnside and the authors themselves, and consequently constant references are made by means of index numbers, which remain the same throughout all these papers.

The conclusions drawn as a result of this extensive study are as follows:

1. In general diagnostic work, exclusive of nervous conditions, a positive reaction is so constant in active syphilis that such a diagnosis, made in the face of a negative result, will require very special support on other grounds before it can be considered probable.

2. A positive reaction in the cerebrospinal fluid indicates a syphilitic lesion of the central nervous system.

3. In active, untreated cases of dementia paralytica, tabes dorsalis, and cerebrospinal syphilis, the reaction is positive both in the serum and cerebrospinal fluid. In cerebral syphilis, without involvement of the spinal cord, the cerebrospinal fluid is usually negative. Except in the latter cases the strength of the reaction has no certain diagnostic value.

4. The reaction is apt to be negative in the serum or cerebrospinal fluid in cases of hemiplegia, non-progressive tabes dorsalis, and old lesions of the nervous system, especially in cases of stationary congenital syphilis. It is also usually negative in the serum particularly in very recent nerve lesions of the late secondary period, and in the cerebrospinal fluid particularly in treated cerebrospinal cases.

5. The occurrence of a positive reaction in one of the test-fluids when the other is negative is so common that a reaction found negative in one fluid only is insufficient to exclude syphilis.

6. Pure "parasyphilitic" lesions do not respond satisfactorily to treatment as shown by the slight effect upon the reaction in the cerebrospinal fluid. Cerebrospinal lesions react very readily to treatment. The celerity with which the reaction in the cerebrospinal fluid responds to treatment is an index of the "parasyphilitic" or "syphilitic" nature of the case.

7. The positive reaction in the serum is rapidly made negative by treatment in acute syphilis; but in chronic syphilis this effect is produced very slowly. In the cerebrospinal fluid, on the other hand, the rate of reduction of the reaction does not depend upon the duration of the infection. Thus, in gummatous lesions of the central nervous system, as also in secondary cerebrospinal syphilis, the effect of treatment upon the reaction in the cerebrospinal fluid is rapid.

8. The "provocative" injection of salvarsan for the purpose of exciting an exacerbation of the reaction has some, but probably an exaggerated diagnostic value.

4. *Bulbar Nuclei*.—Anatomical observations in man and experimental investigations in the lower animals have provided us with an extensive knowledge of the position and relations of the nuclei of the cranial nerves in the medulla oblongata. The author of the paper, which is based upon a case which afforded unusual facilities for investigation, does not claim to do more than confirm our previous knowledge of these nuclei. He does attempt, however, to go further and establish the existence of a salivary center in the medulla oblongata.

The patient, a miner, received a severe injury on the upper part of the neck on April 18, 1913. When a careful examination was made a few days later, the following conditions were observed: (1) Paralysis of the left side of the tongue. (2) Paralysis of the left sterno-mastoid and upper part of the left trapezius. (3) Loss of taste in the whole of the back of the tongue, more marked, however, on the left side. (4) Paralysis of the left side of the palate. No anesthesia of the palate could be detected. (5) Aphonic speech. By laryngoscopic examination the left vocal cord was seen to be immobile. Loss of power of swallowing, even for liquids. In addition to the above symptoms, a slight left facial paralysis was observed on the first day after the accident, but this rapidly disappeared. It is also stated that immediately after the accident some question arose as to whether the phrenic nerve was injured or not, but on the following day no paralysis of the diaphragm could be detected. At no time was there any paralysis of the extremities, nor with the exception of the paralysis of the left sterno-mastoid and trapezius muscles was there any limitation of the movements of the neck.

The author gives a full account of the autopsy findings and results of the microscopical investigations, and summarizes the anatomical changes as follows: (1) Degeneration in the cells of origin of the spinal accessory nerve in the ventro-lateral part of the grey matter of the upper cervical region of the spinal cord. (2) Degeneration throughout the whole region

of the nucleus ambiguus. (3) Partial degeneration of the dorsal nucleus of the vagus. (4) Partial degeneration of the hypoglossal nucleus. (5) Marked fatty changes in the solitary bundle. (6) Scattered degenerated cells in the grey matter of the upper part of the medulla just dorsal to the inferior olive.

There was only a small amount of fatty change present, it being practically confined to the solitary bundle. The length of time which elapsed between the injury and the death of the patient (over two months) is no doubt in part a reason for the limited amount of fatty changes present. Since the dorsal nucleus of the vagus, which is largely a sensory nucleus, showed only partial degeneration, it may be surmised that perhaps the injury to the nerve took place on the distal side of the ganglion, the degenerated cells representing the motor part of this nucleus. In this case, the fatty change in the solitary bundle would represent degenerated fibers from the glosso-pharyngeal nerve. It was, however, impossible to say at exactly what point in their course through the jugular foramen the nerves were injured. From the fact too that the hypoglossal nucleus showed only partial degeneration, we may assume that the injury to the nerve was not complete, a hypothesis borne out by an examination of the skull, for it was found that the fracture did not directly involve the anterior condyloid foramen.

JELLIFFE.

MISCELLANY

A STUDY OF THE METABOLISM OF CALCIUM, MAGNESIUM, SULPHUR, PHOSPHORUS, AND NITROGEN IN ACROMEGALY. Olah Bergeim, F. T. Stewart, P. B. Hawk. (The Journal of Experimental Medicine, Vol. XX, No. 3.)

The mechanisms underlying the metabolism of the mineral constituents of the human body are almost unknown. The present study is a tentative approach to certain aspects of the problems involved. The authors find that in acromegaly a distinct retention of calcium, magnesium and phosphorus was noted, which was not accompanied by corresponding changes in general metabolism, as evidenced by a practical balance of nitrogen and sulphur. It is concluded that there is a primary disturbance in the metabolism of the mineral elements mentioned, with the probable formation of new bony tissue. The distribution of elements between urine and feces shows no abnormal variations. A suggestion is made as to the reasons for variable calcium output in the urine on a uniform diet.

JELLIFFE.

LIVER FUNCTION AS INFLUENCED BY DUCTLESS GLANDS. G. H. Whipple and W. P. Christman. (Jour. of Exp. Medicine, Vol. 20, No. 3, p. 297.)

Physico-chemical metabolism mechanisms are slowly being analyzed with the result that vegetative neurology is opening up as a specialty almost of its own as a fundamental stratum of neurological knowledge. The relation of liver function to certain vegetative neurological structures is here discussed.

The authors find that when phenoltetrachlorophthalein is injected intravenously, it is eliminated from the body in the bile through the activity of the hepatic epithelium. The feces may be collected after purgation and the phthalein extracted and estimated against a standard solution. The

estimation of phthalein can be done with accuracy in a suitable colorimeter and the elimination in normal dogs is quite constant. Given a definite liver injury by means of poisons (chloroform, phosphorus), the amount of phthalein excreted will be diminished and the fall in output will be proportional to the amount of injury. With an acute fatal poisoning the curve may fall to zero. Under certain conditions of vascular interference the liver phthalein may show a decreased output; in passive congestion of the liver and with the Eck fistula the liver output may fall considerably below normal. Known disturbances of the liver function due to parenchymatous injury or vascular disturbances are indicated by a fall in the phthalein excretion curve. Conversely it may be claimed that a drop in phthalein excretion may indicate a decrease in the functional capacity of the liver even if there be no detectable histological changes. Adrenal insufficiency produced by extirpation of three fourths or more of the gland tissue will be associated with a drop in liver phthalein excretion. With hypertrophy of the adrenal fragment the excretion comes back to normal, but may fall again when more adrenal tissue is removed. Pancreatic insufficiency causes a progressive fall in the phthalein excretion indicating a grave lowering of the functional capacity of the liver. This fact has a direct bearing on the question of diabetes. Parathyroid insufficiency with tetany causes no decrease in phthalein output, but at times a rise above normal. This comes out best when the phthalein curve is low following pancreas extirpation. Parathyroid tetany may cause hyperactivity on the part of the liver cells. Thyroid insufficiency produces no change in the uniform curve of phthalein excretion. Hypophysis insufficiency shows an initial fall in the curve, followed by a return to normal and a final drop in the last few days before death. These experiments supply evidence to the effect that the liver is very much concerned in the derangement that follows the removal of the ductless glands. Hence it seems probable that this disturbance of the liver function may be an important factor in the general symptom complex of ductless gland insufficiency.

JELLIFFE.

THE MID BRAIN RESPIRATORY TRACT. Graham Brown (*Journal of Physiology*, 48, 1914, p. 32).

The author says that it has for some time been suggested by Marckwald and others that there exists in the brain of the higher animals a center which in some way regulates respiration. The exact location of such a center has not as yet been forthcoming, but it was supposed to be probably in the *formatio reticularis* at the level of the posterior colliculi. Dr. Brown records an observation which goes strongly to prove the presence of such a respiratory controlling area. The animal used was one of the monkeys. The brain was exposed and divided in the region of the thalamus. On a certain area, to be presently noted, rapid faradic stimulation by the unipolar method resulted in immediate gasping of the animal. There was also rapid panting, and the abdominal muscles contracted and relaxed strongly and rhythmically each second. That these effects were due to the particular site and character of stimulation was shown by the fact that withdrawal of the stimulation caused immediate cessation of the movements. There was also no after-discharge. The area, too, was very limited, and stimulation 1.5 millimeters away from it showed no effect. The site of this "respiratory" area was 3 mm. from the aqueduct, just to the side of it, at a point on the efferent tract (*recto-bulbar?*) of the

anterior corpora quadrigemina. This is an isolated observation, but if confirmation is forthcoming it will add still another nervous center to those already located as responsible for certain coördinated and automatic actions.

PARAMENINGOCOCCUS AND ITS ANTISERUM. Martha Wollstein. (Journal of Experimental Medicine, Vol. XX, No. 3.)

Cases of epidemic meningitis which do not react to serum are often due to a different organism, the parameningococci of Dopter. Wollstein has studied this problem with the following results.

The parameningococci of Dopter are culturally indistinguishable from true or normal meningococci, but serologically they exhibit differences as regards agglutination, opsonization, and complement deviation. Because of the variations and irregularities of serum reactions existing among otherwise normal strains of meningococci it does not seem either possible or desirable to separate the parameningococci into a strictly definite class. It appears desirable to consider them as constituting a special strain among the meningococci not, however, wholly consistent in itself. The distinction in serum reactions between normal and parameningococci are supported by the differences in protective effects of the monovalent immune sera upon infection in guinea pigs and monkeys.

It is therefore concluded that it is highly desirable to employ strains of parameningococcus in the preparation of the usual polyvalent antimeningococcic serum. It remains to be determined whether it is better to employ the parameningococci along with the normal meningococci in immunizing horses, or to employ normal and para strains separately in the immunization process and to combine afterwards, in certain proportions, the sera from the two kinds of immunized horses.

JELLIFFE.

LOCALIZATION OF THE VIRUS AND PATHOGENESIS OF EPIDEMIC POLIOMYELITIS. Simon Flexner and Harold Amoss. (Journal of Experimental Medicine, Vol. XX, No. 3.)

These authors find after considerable experimentation that the virus of poliomyelitis is capable of penetrating the retina without producing apparent injury, to reach the central nervous organs. The virus injected into the blood is deposited promptly in the spleen and bone marrow, but not in the kidneys, spinal cord or brain. Notwithstanding the affinity which the nervous tissues possess for the virus, it is not removed from the blood by the spinal cord and brain until the choroid plexus and the bloodvessels have suffered injury. The intervertebral ganglia remove the virus from the blood earlier than do the spinal cord and brain. An aseptic inflammation produced by an intraspinal injection of horse serum facilitates and insures the passage of the virus to the central nervous organs, and the production of paralysis. The unaided virus, even when present in large amounts, passes inconstantly from the blood to the substance of the spinal cord and brain. When the virus within the blood fails to gain access to the central nervous organs, and to set up paralysis, it is destroyed by the body, in the course of which destruction it undergoes, as a result of the action of the spleen, and perhaps other organs, diminution of virulence. The histological lesions that follow the intravenous injections of the virus in some but not in all cases differ from those which result from intraneural modes of infection. In escaping from the blood into the spinal cord and brain, the

virus causes a lymphatic invasion of the choroid plexus and widespread perivascular infiltration, and from the latter cellular invasions enter the nervous tissues. A similar lymphoid infiltration of the choroid plexus may arise also from an intracerebral injection of the virus. The histological lesions present in the central nervous organs in human cases of poliomyelitis correspond to those that arise from the intraneural method of infection in the monkey. The virus in transit from the blood through the cerebrospinal fluid to the substance of the spinal cord and brain is capable of being neutralized by intraspinal injection of immune serum whereby the production of paralysis is averted. Carmin in a sterile and finely divided state introduced into the meninges and ventricles sets up an aseptic inflammation, but is quickly taken up by cells, including ependymal cells. When an aseptic inflammation has been previously established by means of horse serum, or when the nervous tissues are already injured by the poliomyelitic virus, the pigment appears to enter the ependymal cells more freely. The experiments described support the view that infection in epidemic poliomyelitis in man is local and neural, and by way of the lymphatics, and not general and by way of the blood. Hence they uphold the belief that the entrance of the infection is the upper respiratory mucous membrane.

JELLIFFE.

FURTHER STUDIES ON THE DEVELOPMENT OF THE CRANIAL SYMPATHETIC GANGLIA. Albert Kuntz. (The Journal of Comparative Neurology, Vol. 24, No. 3.)

Following an extended investigation of the development of the sympathetic nervous system in the vertebrate series, in which the sympathetic ganglia in the cranial region were not specifically considered, the writer has undertaken an investigation of the development of the cranial sympathetic ganglia in types of the several classes of vertebrates. Observations on the development of these ganglia in embryos of the pig have been published in an earlier paper by the same author. The present paper embodies the results of a comparative study of the development of the cranial portion of the sympathetic nervous system in embryos of the common toad fish (*Opsanus tau*), the frog and *Ambystoma*, the turtle, the chick, and the pig. In the general investigation of the development of the sympathetic nervous system, with which are associated the names of not a few able investigators, the cranial portion of that system has been quite generally neglected. Observations on the development of the ciliary ganglion in types of the several classes of vertebrates have been recorded by not a few investigators, many of whom studied the development of this ganglion in connection with their investigations of the development of the eye-muscle nerves. Recorded observations on the development of the remaining sympathetic ganglia in the cranial region, in the lower vertebrates, are fragmentary and incomplete.

The author has not attempted a systematic review of the literature bearing on the development of the cranial portion of the sympathetic nervous system. For a more or less complete review of the literature on the development of the ciliary ganglion in types of the several classes of vertebrates he refers to Carpenter's paper on "The Development of the Oculomotor Nerve, the Ciliary Ganglion, and the Abducens Nerve in the Chick."

In his discussion and conclusions he states that according to his observations the cranial sympathetic ganglia bear the same genetic relationships to the cerebrospinal nervous system, in all the classes of vertebrates, as do

the ganglia of the other parts of the sympathetic nervous system, *i. e.*, they arise from cells which have their origin in the cerebrospinal ganglia and the wall of the neural tube and advance peripherally along sensory and motor nerve roots respectively. Not all the nervous elements taking part in the development of the sympathetic ganglia actually migrate as such from the cerebrospinal ganglia or the neural tube; many of them arise from the mitotic division of cells which have advanced peripherally from the cerebrospinal nervous system. The character and destiny of the cells which become separated from the cerebrospinal nervous system and advance peripherally along the cranial and spinal nerves have been discussed in earlier papers by the author and are not, therefore, considered in the subject of this abstract. The majority of these elements are cells of the "indifferent" type, many of which retain the capacity for cell division after they have become separated from the cerebrospinal ganglia or the neural tube. Consequently, mitotic figures may be frequently observed along the paths of migration and in the peripheral ganglia. Such mitotic division of nervous elements in the paths of peripheral nerves and the primordia of peripheral ganglia probably occurs less frequently in embryos of the lower than in embryos of the higher vertebrates. Neither do all the elements arising in this manner take part in the development of the sympathetic ganglia. Many of them obviously become differentiated into cells of the neurilemma.

The distribution and the relative degree of development of the cranial sympathetic ganglia vary greatly in the several classes of vertebrates. The degree of development of the several cranial sympathetic ganglia is obviously correlated with the demands of the functions of the structures innervated by the sympathetic nerves associated with them.

The author sums up as follows: (1) The sympathetic ganglia on the cranial portion of the nerve trunks arise, in embryos of the toad fish, primarily from cells derived directly from the first spinal ganglion and the cerebral ganglia associated with the X, VII and V cranial nerves. Certain of these ganglia receive cells also which advance peripherally from the neural tube along motor nerve-roots. The ciliary ganglion arises in the path of the oculomotor nerve primarily from cells which advance peripherally from the mesencephalon along this nerve. It later receives a relatively small number of cells which advance peripherally from the Gasserian ganglion and the first sympathetic ganglion associated with the latter along the radix ciliaris longa. (2) A permanent ciliary ganglion is probably not developed in *Amblystoma*. In larvæ of the frog, the ciliary ganglion arises in essentially the same manner as in embryos of the fish. Other distinct cranial sympathetic ganglia probably do not occur in the Amphibia. (3) In embryos of the turtle, the ciliary ganglion arises in the path of the oculomotor nerve primarily from cells which advance peripherally from the mesencephalon along this nerve. After this ganglion has become connected with the ophthalmic nerve it receives a relatively small number of cells which advance peripherally from the Gasserian ganglion. The sphenopalatine ganglion arises, in embryos of the turtle, in the paths of the great superficial petrosal nerve and soon becomes connected by fibrous rami with the maxillary nerve. It arises from cells which advance peripherally from the geniculate and the Gasserian ganglia respectively along the great superficial petrosal and maxillary nerves. Ganglia homologous with the otic and the submaxillary ganglia of the higher vertebrates were not observed

in embryos of the turtle. (4) In embryos of the chick, the ciliary ganglion bears the same genetic relationships to the oculomotor and the ophthalmic nerves and arises in essentially the same manner as in embryos of the turtle. The otic ganglion arises, in embryos of the chick, in the path of a tract of sympathetic fibers which emerge from the sympathetic plexus surrounding the carotid artery, primarily from cells which are derived from the superior cervical and the geniculate ganglia. The sphenopalatine ganglion arises, in embryos of the chick, primarily from cells which advance peripherally from the geniculate ganglion along the great superficial petrosal nerve, but probably receives cells also which advance peripherally from the Gasserian ganglion along the maxillary nerve. The relatively small submaxillary ganglion is genetically related to the mandibular nerve. (5). In embryos of the pig, the ciliary ganglion is genetically related to the oculomotor and the ophthalmic nerves. The sphenopalatine ganglion arises from cells which advance peripherally from the Gasserian ganglion along the maxillary nerve. The otic and the submaxillary ganglia are genetically related to the mandibular division of the trigeminal nerve and probably receive cells from both the Gasserian ganglion and the wall of the rhombencephalon.

JELLIFFE.

Notes and News

The Massachusetts Society for Mental Hygiene has opened an office at Room 313, Ford Building, 15 Ashburton Place, Boston. The officers of the Society are: the Honorable Harvey H. Baker, Boston, President; Doctor Walter E. Fernald, Waverly, Vice President; Doctor Charles E. Thompson, Gardner, Secretary; John Koren, Esquire, Boston, Treasurer; Executive Committee: Miss Edith M. Burleigh, Boston; Doctor James J. Putnam, Boston; Doctor Alfred E. P. Rockwell, Worcester; Doctor Henry R. Stedman, Brookline; Professor Robert M. Yerkes, Cambridge. Doctor Frankwood E. Williams, formerly Resident Physician at the Psychopathic Hospital, Ann Arbor, Michigan, and first Assistant Physician at the Psychopathic Hospital, Boston, has been appointed Executive Secretary.

The Journal OF Nervous and Mental Disease

An American Monthly Journal of Neurology and Psychiatry, Founded in 1874

Original Articles

UNILATERAL HYPERTROPHY OR TROPHEDEMA OF LIMBS. REPORT OF A CASE¹

BY HOWELL T. PERSHING, M.D.

DENVER

In June, 1913, I was consulted by Miss H., aged 19, on account of the right limbs being larger than the left. She has since remained under observation and the conditions presented are so rare that they should be recorded.

Family History.—The father is now 51 and in good health. Father's father died at 66 after a four months' illness in which "asthma" was the principal symptom, his previous health having been good. Father's mother died at 70; she was always of feeble vitality and had two operations for epithelioma of the face. All of the father's seven brothers and sisters still living are described as not very strong; two died in infancy.

The mother is now 48. She has a mitral regurgitant murmur with good compensation. In 1901, a few months after a severe fright, she began to have paroxysms of pain in the fingers with heat and redness followed by cyanosis. I treated her for this condition, regarded as Raynaud's disease in 1905. In 1907 necrosis of the terminal joint of the left thumb, succeeding a series of these attacks, required amputation. The mother's father died at 72; his health before the last illness was good. The mother's mother died at 52, probably of strangulated hernia. One of the mother's sisters is nervous; two brothers and two sisters are well.

¹Read at the fortieth annual meeting of the American Neurological Association, May 7, 8 and 9, 1914.

The patient's older brother, now well, had migraine in childhood, a younger sister suffers from constant backache, a brother aged 8 is described as not strong. No member of the family is known to have had anything like a localized hypertrophy or atrophy.

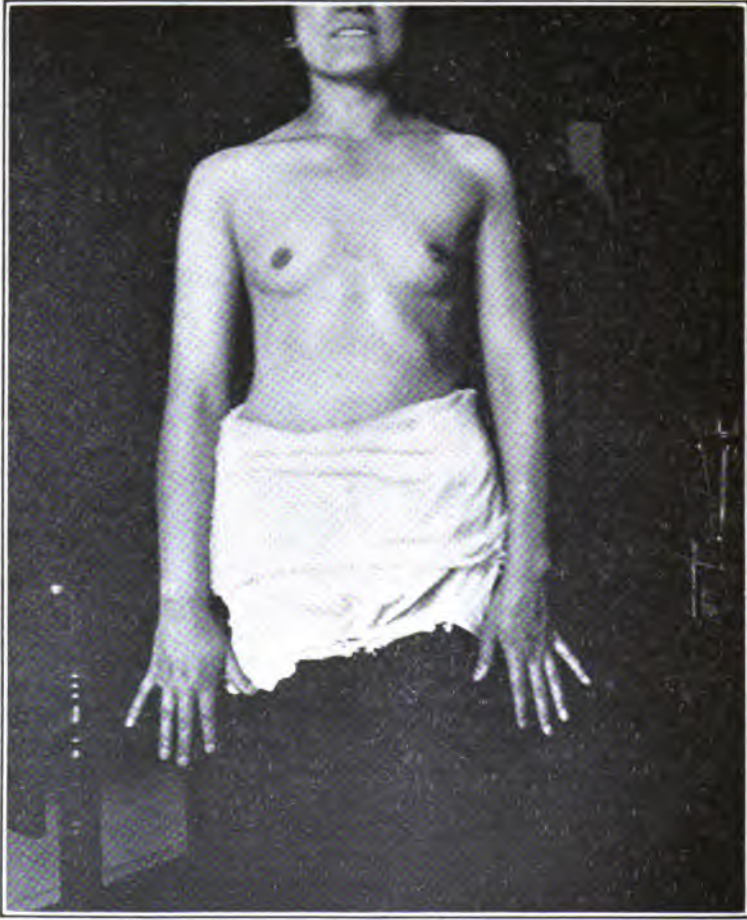


FIG. 1. Note greater thickness of arm and forearm on the right side.

The patient herself had a normal birth and developed normally until 3 years of age when she had a severe illness supposed to be typhoid fever.

After recovery from this, the left limbs were for the first time noted as smaller than the right, and they were specially exercised.

to bring their development up to the supposedly normal right ones. At 7 years there was an attack of scarlatina with good recovery. At 13 menstruation was established and has since been quite regular and normal. Typical attacks of migraine, which had recurred at times since childhood, became very frequent during the winter of 1911-1912 when she was working as a telephone operator.

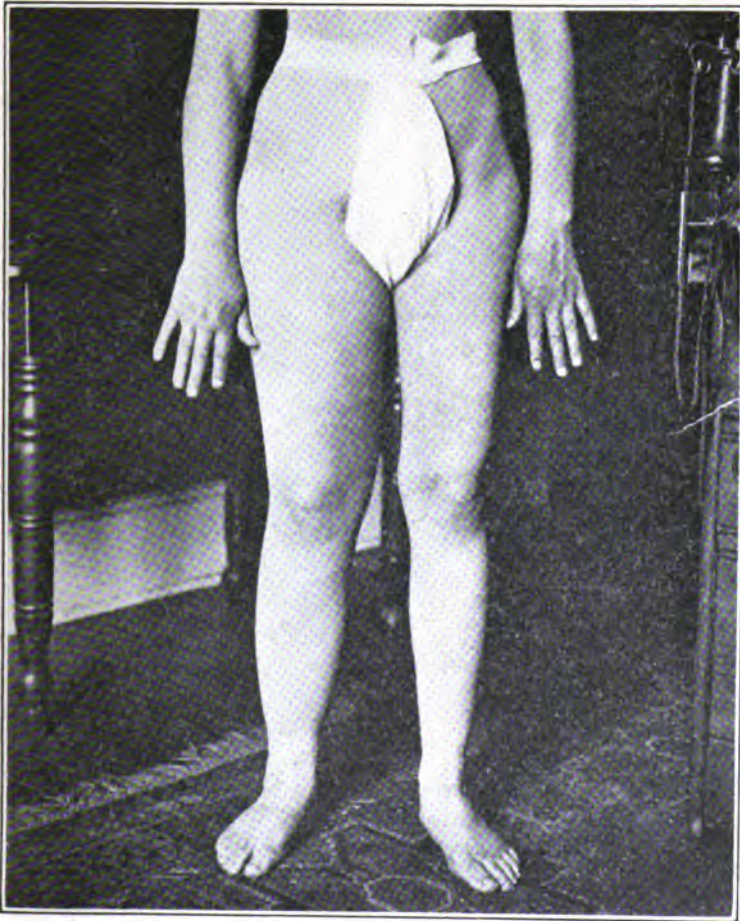


FIG. 2. Showing greater thickness of thigh and leg on right side without increase in length and with very little difference in the feet.

Once in the summer of 1913, while taking thyroids, the right index finger became blanched, suggesting the onset of Raynaud's disease, but circulation returned and the symptom has not recurred.

The difference in the size of the limbs on the two sides gradually increased from the age of 3 and the increase was especially rapid during the 14th year when menstruation was established. It then became apparent that the right limbs were abnormally large.

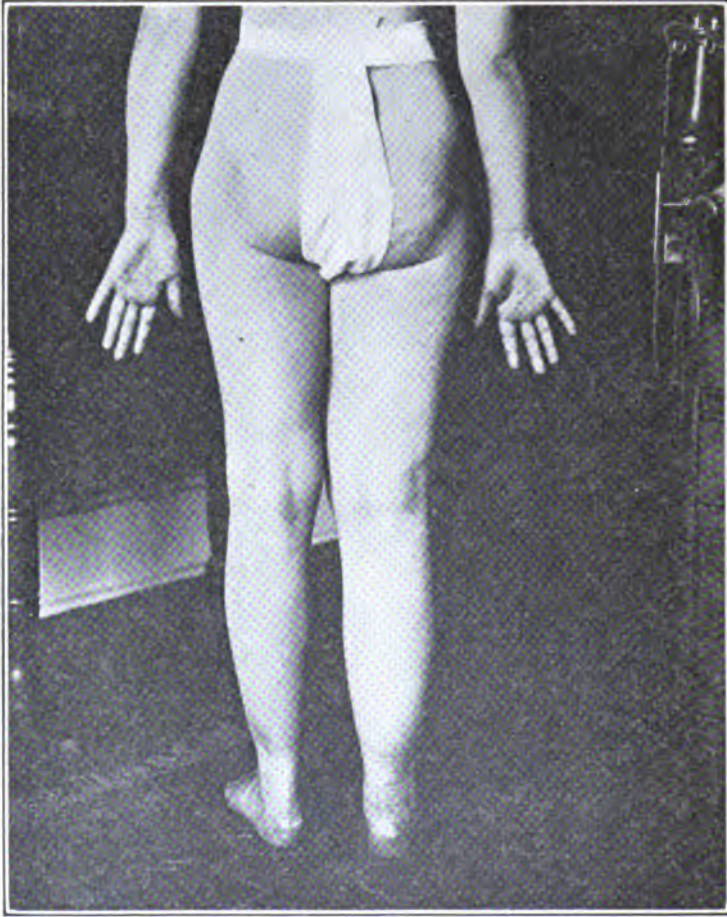


FIG. 3. Showing same relations as Fig. 2.

The hair of the head has been rather scant, harsh and dry, splitting easily, and has seemed to grow longer on the *left* side. The pubic and axillary hair has also developed rather scantily but otherwise normally. The voice has been low pitched and slightly husky; she has been unable to sing, especially the higher notes. There has been a heavy feeling in the throat and she has habit-

ually tried to clear it by little coughs. On exertion she soon tires all over. Family and school life and mental development have been entirely normal.

On examination no motor or sensory loss or change in the reflexes could be detected. Dissociation of the forms of sensation was especially looked for, but not found. The heart was normal, blood pressure 120, the same on the two sides. Hemoglobin, red cell count and differential leucocyte count normal. The skin showed no abnormality except undue moisture especially



Right.

Left.

FIG. 4. The right hand is very slightly larger than the left.

of the hands and axillæ. The patient thinks perspiration is greater on the right side of the body and is sure that the right arm does not bleed as readily as the left. The urine was normal.

Inspection and palpation of the neck with especial reference to the thyroid gland detected nothing abnormal.

The difference in the bulk of the limbs on the two sides is well shown in the photographs and in the following measurements:

Circumference of	Right	Left
Thigh	54 cm.	48 cm.
Calf	39	32
Arm	25	22.5
Forearm	25	23
Hand	18.8	18.3

In length the lower limbs are equal. The right arm from acromion to tip of middle finger is 73 cm.; the left 72.3 cm.; a barely measurable difference.

The head, neck and chest are symmetrical. The abdomen is nearly symmetrical, but just above the right groin and right iliac crest the subcutaneous tissue is somewhat thicker than on the left side. Passing upward this difference steadily diminishes and at the margin of the ribs, the thickness on the two sides is the same.



Left.

Right.

FIG. 5. The bones and muscles are alike on the two sides, but on the right the subcutaneous tissue is very greatly increased.

The subcutaneous tissue, estimated by picking up a fold, is obviously much thicker in the right limbs than in the left, the difference in this tissue alone apparently accounting for the asymmetry. This observation is confirmed by the radiographs, which show no difference in the size of the bones, right and left, a little in the muscles as a whole, probably accounted for by the inter-muscular tissue, and a very great difference in the thickness of the subcutaneous tissue.*

*A very slight difference in the shadows of the bones is fully accounted for by the bones on the right side being brought slightly nearer to the source of the rays by the thickened soft parts.

This tissue is firm and does not pit on ordinary pressure, but prolonged pressure makes an indentation which persists for a considerable time. The indentation made by the shoe top is apparent in the photographs. There is no impairment of the general health, except a certain amount of worry and consequent depression and irritability caused by the knowledge of the deformity.

This case is clearly related to the hereditary edema of Milroy¹ although sharply contrasted with this eminently familial group in affecting only one member of the family. It corresponds exactly to a group of cases reported together as trophedema or hypertrophy by Meige,² Rapin,³ Hertoghe⁴ and Mabile.⁵ Meige defines the condition as follows: "An edema which is chronic, pale, firm, painless, of segmentary distribution, unilateral or bilateral, isolated or familial and hereditary, sometimes perhaps also congenital."

Little can be said as to causation. In the hereditary cases both the original cause and the mechanism of inheritance are unknown. In other cases as well as in this one the onset has followed one of the infectious fevers such as typhoid, measles, smallpox or scarlet fever, but the possibility of the relation being merely one of coincidence is obvious. Rapin speaks of the disorder as myelopathic but signs of organic disease of the cord are lacking in all the cases. One naturally speculates as to functional derangement of trophic and vasomotor impulses and as to disorder of the ductless glands.

On first seeing the patient, with the mother's history in mind as suggesting a relative insufficiency of the thyroid and taking into account the character of the thickened subcutaneous tissue, the harshness of the hair and huskiness of the voice, I made a provisional diagnosis of myxedema, in spite of the apparently normal condition of the thyroid and the fact I had never heard of a unilateral myxedema. The failure of careful persistent thyroid medication to do any good, caused this diagnosis to be abandoned, but I note with interest that the French and Swiss observers made the same diagnosis of thyroid insufficiency and Hertoghe would not give it up in spite of the failure of thyroid medication.

The only treatment so far known that has any certain effect is the application of elastic bandages. This of course is only palliative but it is worth while. The fact that in my patient the right

foot is but little larger than the left, is no doubt due to the restraining pressure of the shoe.

This is called a case of unilateral hypertrophy because such cases have usually been reported under the general head of hypertrophy.

Meige's term, trophedema, is more precise, but Rapin and Mabile, in the same number of the *Nouvelle Iconographie*, use the terms hypertrophy and hemi-hypertrophy in their titles. Moreover, trophedema is really an hypertrophy if only of the connective tissue.

The clavicles in his own case were alike. True, one of the photographs makes the inner end of the right clavicle seem enlarged but this is a deceptive effect of shading.

1. Milroy. An Undescribed Variety of Hereditary Edema. *N. Y. Med. Jour.* 1892.
2. Meige. Sur le trophoedème. *Nouvelle Iconographie de la Salpêtrière*, 1901, 6.
3. Rapin. Sur une forme d'hypertrophie des membres. *Ibid.*
4. Hertoghe. Contribution a l'étude du trophoedème chronique. *Ibid.*
5. Mabile. Observation de trophoedème. Deux cas d'hémihypertrophie congénitale du corps. *Ibid.*

LOSS OF MEMORY ALLEGED BY MURDERERS

BY THEODORE DILLER, M.D.

PITTSBURGH

In an interesting communication before the last meeting of the American Neurological Association, Dr. George Walton, basing his experience on a large number of cases of which he had knowledge, pointed out that very commonly persons accused of murder denied all memory of the actual killing, although they might remember events up to within a very short period of the act and very shortly afterwards. I believe there can be no doubt whatever that this statement is correct, and this view was borne out by testimony of those who discussed Dr. Walton's paper. Certainly this is my own experience. In examination of a considerable number of persons charged with murder, denial of actual killing was made in almost every case.

The discussion of Dr. Walton's paper chiefly turned on the question as to whether this loss of memory of the actual murder was real or feigned; it was pointed out by two or three of the speakers that we could not be perfectly certain in any given case that the amnesia was real or that it was feigned. However, as for myself, I have been disposed to regard this loss of memory as real and not feigned; and for these reasons: (1) It is unlikely that the memory of the killing would be feigned so very *generally* by persons who committed murder; (2) The fact that repeated questioning and cross-questioning, threats of all sorts have failed to shake the statement made by murderers as regards this point when generally they seem so communicative about everything else.

I have offered to myself the following reason for this loss of memory: When the murder is committed it is done at the height of great emotional disturbance; and because of the great intensity of emotion the act, the thoughts which occur at that time are not registered in the memory as the photographic film upon which an intense light is thrown fails to register a picture and only a blur

is produced. To illustrate, I might mention it often happens that persons who are subject to violent attacks of anger retain no memory whatever of words uttered or conduct performed during such a paroxysm.

In this connection, I wish to make mention of two murder cases which came under my observation, one four years ago and one only last year. These were both cases of what I took to be paranoid dementia præcox; both persons were acquitted of murder by reason of insanity and sent to Dixmont Asylum near Pittsburgh where they have remained up to the present time. After reading the abstract of Dr. Walton's paper and the discussion thereon, I went down to Dixmont, two days ago, and by the courtesy of Dr. Hutchinson, the superintendent and Dr. Barrett the assistant physician, reexamined both these men. Both of them denied all memory of the actual murder, just as they had when I examined them in jail before the trial. I pointed out to both of them that their cases were settled and no harm could come to them by an admission of memory of the deed and tried in every possible way to ascertain that their loss of memory was shammed. But in both cases denial of the memory of the killing was complete and made in precisely the same terms as before the trial; and their manner showed no real concern for the act which they had committed and each retained the same delusions he had entertained before the murder.

So here are two cases in which events point very strongly to the conclusion that the loss of memory for the murder was real.

THE NEUROPATHOLOGICAL FINDINGS IN A CASE OF PERNICIOUS ANEMIA WITH PSYCHICAL IMPLICATION

By J. A. F. PFEIFFER, M.D.

GOVERNMENT HOSPITAL FOR THE INSANE, WASHINGTON, D. C.

Severe nervous symptoms are a frequent clinical entity in pernicious anemia, and although mentality is rarely impaired, psychical phenomena of varying intensity have been observed.

It is therefore pertinent that this fact should be appreciated, especially the occurrence of psychic derangement in the initial stages of the affection, as obviously not a few cases of this character fail to be diagnosed early in consequence of such symptoms, which tend to obscure the true nature of the disease process.

The object of this paper, however, is to consider chiefly the histopathological alterations involving the brain and spinal cord in a case of pernicious anemia with nervous and mental symptoms, since little is known regarding the cerebral lesions, and apparently but few attempts have been made to study the anatomical changes in the brain from this disease.

C. J., aged 55, was admitted to the Government Hospital for Insane, on June 15, 1911.

Family History.—Father died at the age of forty-eight from typhoid fever. He had always been a healthy man of good habits. Mother is living and enjoys health at the age of seventy-four years. The patient was the eldest child of a family consisting of one girl and two boys. At the time of writing both his sister and brother are alive and healthy. There was no history of nervous or mental diseases in the family, and no history of syphilis or tuberculosis.

Personal History.—The patient when a child was healthy and well nourished. He had whooping cough at the age of nine years, but otherwise never suffered from any serious illness at any time. He went to school at the age of six and was considered a good scholar. He continued at school for about nine years, leaving when sixteen, and began work on a farm, where he remained for several years. He was married when twenty-two years of age,

and then immigrated to southwestern Nebraska, where he secured a homestead claim, and after a residence there of three years, acquired a clear title of ownership in the property. Subsequently he sold his farm at a considerable profit and obtained a position in a general store. After holding this position for several years, he decided to become a veterinarian, and entered a veterinary college, from which he was graduated at the end of a course extending over a period of two years. He practiced his profession for eleven years, and later became an inspector. He never had any venereal disease and did not indulge in the use of alcohol or drugs.

History of Present Illness.—In February, 1911, several months before his admittance to this hospital, the patient suffered with fatigue and nervousness. Digestive derangements were in evidence, and an indisposition to take food, associated with nausea occurred. No symptoms referable to the nervous system were present at this time, except persistent headache, a creepy feeling over his entire body and paroxysmal sensations of warmth. Owing to weakness he was compelled to remain at home, but after some days his condition improved sufficiently for him to resume his usual occupation. In April of the same year he was required to move to Texas. During the first few weeks of his residence there, he seemed fairly well, and was vaccinated against typhoid fever without any unusual manifestations. Some weeks later, however, he again experienced symptoms of languor, progressive weakness, continuous headache and intestinal disorder, in consequence of which he entered a hospital. While there he became delirious. On admission to this hospital the patient presented a fairly well nourished appearance, although there was evidence of some emaciation. The skin was of a slight yellow tint and the mucous membranes were pale. The respiratory system showed no signs of pulmonary disease, but the cardiac impulse was feeble. There were some vomiting and tenderness of the abdomen. The abdominal organs were normal, as far as could be determined. No sclerosis of the peripheral vessels was detectable, and the blood pressure was normal. He was weak and although exceedingly restless, made no attempt to arise from his bed. He frequently moved his arms about in a rather aimless manner, and eventually showed evidence of considerable confusion. The temperature was slightly subnormal when admitted. On several occasions, however, it rose to 101° F., but for the most part was normal throughout the entire course of his illness. His pulse was usually rather feeble and varied between 75 and 90 beats per minute. Urinalysis showed nothing abnormal, and examination of the feces was negative. A blood examination was made shortly after admission with the following results: Red blood cells, 900,000; hemoglobin, 30 per cent.; color index, 1.6; white blood cells, 6,000; polynuclear neutrophils, 57 per cent.; lymphocytes, 36 per cent.; normoblasts were encountered and several megaloblasts.

A systematic microscopic examination of the blood was instituted, and showed the characteristic feature of pernicious anemia. Normoblasts were invariably present, and sometimes occurred in considerable numbers. Megaloblasts were usually in evidence. The lymphocyte count was highest on November 30, reaching 40 per cent.; the polynuclear neutrophile count was 50 per cent. The Wassermann test with both the blood and spinal fluid was negative.

The following clinical data indicate the condition of the patient during his residence at this hospital.

A few days after his entrance some improvement was observed, but he was irrational at times and suffered with drowsiness and persistent headache. Nausea and vomiting continued. He took but little nourishment and considerable dyspnea was in evidence.

His condition gradually improved and by July 10 the nausea and vomiting had ceased and his appetite was better. His headache had disappeared and only slight dyspnea was apparent. At this time he asserted that he had no recollection of what had transpired while in the hospital. He complained of stinging and tingling sensations in his arm and legs which he stated were due to electric currents traversing his bed. Various auditory hallucinations were present. Both the deep and superficial reflexes were easily obtained.

No marked variation in his condition occurred in the following month. Paresthetic sensory disturbances continued, and he asserted that the mattress became very hot; occasionally he was allowed to be up and about, but on account of his weakness it was necessary for him to return to bed after a short interval. Delusions of a mild persecutory character were present and brief periods of forgetfulness.

On September 3, his temperature rose to 101° F. and there was pronounced physical discomfort. Considerable gastro-intestinal disorder was associated with this illness and vomiting spells occurred. He was extremely weakened. His enunciation was defective and speech was slightly thickened. The mucous membranes were anemic and the skin of a distinct lemon-yellow color. He promptly recovered from this attack, however, and subsequently showed marked improvement, notably regarding his mental status. During the month of October and early part of November no special change in his physical condition was observed, but his intellect frequently appeared quite normal and he responded well to sensory tests. Some interesting changes were discovered upon examination of the nervous system, and following is a summary of the results secured during this period.

The cranial nerves were normal. The pupils were equal and regular in outline. Reacted to both light and accommodation. By ophthalmoscopic examination the vessels appeared small and the discs pale. No defectiveness in the movements of the eyes or face was evident.

The muscles of the extremities were poorly developed, but retained their contours. No deficiency in power could be detected, but ataxia was well marked. The FN, FF and FT tests could not be performed. His gait was ataxic and Romberg's sign was present. The tendon reflexes of the upper extremities were readily demonstrated.

Tactile sensibility was not affected. He responded quickly and excessively to pin pricks, the hyperesthesia being more pronounced in the abdominal region and lower limbs. Heat and cold were differentiated without difficulty. Some disturbance in the appreciation of passive position was apparent. He was able to discriminate gross differences in weights, but failed to detect slight variations.

In the latter part of November his condition grew worse. Asthenia was marked. He was unable to go about without assistance, and owing to a tendency toward syncopal attacks he was confined to his bed. Restlessness and hallucinations were prominent. Vomiting and diarrhea were not infrequent.

A progressive failure in his physical condition occurred in spite of treatment and he died on December 29.

POSTMORTEM RECORD

External Examination.—The autopsy was performed four hours after death, and the body was in an excellent state of preservation. The body was considerably emaciated, and the skin presented a uniform lemon-yellow tint. The thyroid and lymphatic glands were not enlarged. The teeth were in good condition, but the gums were slightly inflamed, and the tongue furred. No skin eruptions, hemorrhages, edema, or scars were in evidence.

Internal Examination.—Thorax: The superficial fat was greatly decreased, and the muscles appeared dark red in color. The pericardium was unaltered, and the fluid which it contained was clear and normal in quantity. The heart weighed 50 grams. It was soft and flaccid in consistency. The right auricular wall was thinned, and the cavity dilated. The tricuspid orifice admitted five fingers; the leaflets were normal. The right ventricle was enlarged and its wall diminished in thickness. The chambers of the right side of the heart were also dilated. The mitral orifice admitted four fingers; the leaflets were thickened. The aortic valve was well preserved.

The lungs were edematous, somewhat emphysematous and slightly pigmented. The bronchial and mediastinal glands showed nothing peculiar.

Abdomen.—No ascites or peritoneal adhesions were in evidence, but the mesenteric and peritoneal lymph glands were slightly enlarged.

Liver.—Weight 1,700 grams. The surface of the organ was

smooth, and the capsule slightly thickened. It was of a brownish yellow and soft in consistency. The Prussian blue reaction for iron was readily obtained. The gall bladder contained about 20 c.c. of dark yellow colored bile.

The spleen weighed 350 grams. It was slightly enlarged, and the capsule somewhat thickened. The pulp was soft and dark red in color. The macroscopic reaction for free iron was positive. The pancreas appeared normal.

The stomach was slightly dilated and atrophy of the mucous membrane in evidence. The intestines were similarly dilated and the mucosa atrophic.

Genito-Urinary Tract.—The right kidney weighed 220 grams, and the left 250 grams. Both kidneys were slightly enlarged. The capsules stripped readily, the surfaces were smooth and of a grayish-white color. On section the cortices were somewhat thickened and of a uniform pallor, similar to that of the surface. The medullary portions were of a dull red color. The bladder and prostate showed no abnormalities.

The bone marrow presented a dark red appearance, and microscopically hyperplasia was in evidence.

Head.—The calvarium was symmetrical and the wall thinned. The dura was not adherent, thickened or discolored. The cerebrospinal fluid was slightly increased. The pia in general seemed normal, although some opacity was discernible over the base of the brain.

Brain.—Weight 1370 grams. The brain appeared somewhat edematous, but the convolutional configuration was good. The basal vessels were well preserved and no abnormalities were perceptible. The lateral ventricles were not dilated, and the ependymitis was absent. Some of the cerebral veins were slightly distended.

Spinal Cord.—The membranes of the cord were not visibly altered, and the spinal nerves seemed in good condition. The cord was firm throughout and no deformities, shrinkage or softening could be detected. Upon section, however, the posterior and lateral columns were obviously darker than normal.

MICROSCOPICAL STUDY

Pieces from various regions of the brain and spinal cord were treated with the following reagents: Formol, alcohol, Müller's fluid, Weigert's mordant and Orth's fluid.

Different methods of staining were utilized, viz.: Nissl's, Mallory's, Mann-Alzheimer, Van Gieson's, Marchi's, Weigert's, Herxheimer's, toluidin blue, cresyl violet, thionin, etc.

Examination of sections from the cervical and dorsal cord by the Marchi method shows definite degeneration in the posterior columns, and a light-grade diffuse alteration is visible in the lat-

eral tracts. In the lumbar region a small flask-shaped area of degeneration is present in the center of each posterior column.

Histologically round, oval or irregular aggregations of black or grayish-black masses are perceptible. A nucleus is usually seen lying among the granules and surrounded by a free area. Many of the aggregations are composed of fine regular particles; others of small round symmetrical drops, while others contain a large mass encircled by a few smaller granules. Groups consisting of large masses only are encountered, and here and there ring-like formations are visible most frequently around the periphery of the fatty aggregations. These formations are not infrequently yellowish in color, and granules of a similar tinge are encountered apparently imbedded within the large dark masses. Lying within the perivascular spaces fat droplets of various sizes and shapes are seen. These are distributed in irregular quantities along the course of the vessel. The large black particles predominate in the anterior part near the central canal; only a few fatty drops being in evidence. In the outer part of the zone of degeneration groups of regularly shaped small round drops are perceptible. The median zone contains aggregations of granules and fine particles intermingled. Masses surrounded by fat drops, are also encountered.

In longitudinal sections the products of degeneration appear as rows and groups of granules. It is readily discernible in preparations which have been counterstained that the disintegration products are for the most part within the cytoplasm of glia cells. Large proliferating glia cells construct a thick network between the disintegrated nerve fibers.

The area of degeneration does not present as uniform an appearance in sections stained with the hematoxylin-van Gieson method. Considerable variation in the microscopical picture exists in different parts of the area. In the anterior portion and along the borders of the zone the glia net appears distended, and within the dilated meshes swollen medullary sheaths and axis cylinders are seen; and occasionally normal nerve fibers are encountered. The proliferated glial processes usually appear homogeneous. The cells, in cross section, are spider-like in character and contain one or more large pale swollen nuclei with granular nucleoli. The cytoplasm around the nuclei is indistinct, or frothy. In many places, especially towards the median part of the area of degeneration, a granular disintegration or rarefaction of the homogeneous glia substance is perceptible, and in other instances a syncytial structure is visible. The reticulum is seen to envelop groups of nuclei and the glia septa between the particles of detritus. In general, along the borders of the glia network where products of degeneration are abundant, crescent-shaped Körnchencells are numerous. Körnchencells of various sizes and shapes are likewise frequent, and massive stellate glia cells with extensive processes

are encountered. Large Körnchencells containing vacuoles, myelin and axis-cylinder rests are in evidence.

In the periphery of the area a number of cells are seen representing an early stage in the development of the Körnchencells. They have become rounded in contour, lost their delicate processes and lie free within the reticular structure. The majority, however, still retain their attachment to the glia by slender protoplasmic threads. A few irregularly shaped Körnchencells are also observed with an exceedingly coarse cytoplasmic network, and containing many spherical particles of myelin. The nuclei deviate considerably in their appearance, but in newly developed Körnchencells are large, round and pale, or sometimes oval with a fine granular net-like nucleoplasm and nucleoli. Cells which show regressive nuclear changes are perceptible. The nuclei of these cells are notched, shrunken or indicated only by a few remaining granules. The greatest degree of glial proliferation obtains in the median part of the area. Here the large indented stellate cells with distended irregular nuclei predominate, but small cells are also in evidence. Many scavenger cells are seen lying within the meshwork surrounding the blood vessels.

With the Weigert and Weigert-Pal myelin-sheath stains, the zone of degeneration assumes similar dimensions to that described by other methods. Closer study discloses comparatively few darkly tinged particles in the periphery of the area, but toward the inner part they are abundant. Körnchencells and their relationship to the reticulum is readily discernible. The cells with fine net-like cytoplasm rarely contain granules, but those which exhibit coarse meshwork are usually filled with products of disintegration.

With Sudan III stain numerous Körnchencells filled with fat are seen in the area of degeneration. The cells are either round or oval, mostly filled with red colored drops, and possess one or more nuclei, which usually appear shrunken. In longitudinal sections the cells are collected into rows of irregular length. Some are completely filled with fat, while others contain an inconsiderable amount. Occasionally large dark red masses are seen, having numerous smaller granules in their vicinity.

By the Mallory aniline blue method a network is visible in the degenerated region which appears to proceed from the glia cells, occasionally large vacuoles are perceptible in these cells. The nuclei are distorted and pressed towards the periphery of the cell body by the fat vacuoles, and are surrounded by only a small rim of protoplasm. Numerous thin fiber-like processes are seen connecting the cytoplasm of different glia cells. The Körnchencells are of various sizes and shapes, and show considerable diversity in their structure. The meshwork may be uneven and relatively thick, or in other cells fine, small and circular in character. Delicate prolongations are perceptible radiating from these cells into the neighboring tissue. The cytoplasm is occasionally collected only

at the poles of the nucleus giving the cell an elongated aspect. Many of these glia cells show an unmistakable tendency to become rounded in contour, especially those in which the cell body no longer appears homogeneous or the meshwork regular. The processes of these cells gradually disappear, the remaining protoplasm in the vicinity of the nucleus vanishes and is replaced by a network, and the nucleus becomes notched and shrunken. It is evident that these cells become separated from the surrounding syncytium and transport products of disintegration from the ectodermal tissue to the mesodermal vascular system, constituting the most constant and characteristic type of gliogenetic scavenger cell. Quiet frequently large fat vacuoles are seen lying within the cytoplasm of glia cells which possess unusually prominent processes. Such processes not infrequently terminate in the walls of capillaries as triangular shaped formations, or merge into the neighboring tissue. The type of Körnchencells under description is found in considerable numbers in the region of blood vessels associated with a filamentous structure apparently of glial origin. The changes observed in the vicinity of the capillaries are interesting. Some cells are seen which present a fringed aspect, resulting from the singular relationship between the filamentous processes and the network of cytoplasm. Considerable diversity is exhibited in the number and size of the meshes, the form of nuclei, and the thickness of the processes. These cells obviously represent transitional types of Körnchencells. It would appear that the fat contained within many of the gliogenous Körnchencells is reabsorbed by other cells, and the former are eventually restored, and resume their function as supporting elements of the nervous system.

The nerve cells of the gray matter were carefully examined at various levels of the cord by the Nissl stain. With this method the area of degeneration in the posterior columns is also quite perceptible, and the cellular changes readily discernible.

The majority of the anterior horn cells in both the cervical and thoracic regions show no obvious alteration. In some of them, however, evidences of a degenerative process is visible. A number of these cells appear shrunken, elongated and stained more deeply than usual. In such cells the nucleus is, as a rule, unaltered. The pigment in a few instances is abnormally increased, other cells are somewhat swollen, their nuclei eccentrically situated, and chromatolytic changes present. The alterations are more intense in the cells of the posterior horns. Some of the cells are swollen, their outlines are very irregular and complete disintegration of the tigroid substance has occurred. The nucleus is distended and the nucleolus stains poorly. In other cells the nucleus is sometimes shrunken and difficult to distinguish from the cytoplasm owing to the fact that it stains uniformly throughout. A number of the cells of the lateral horns are considerably altered, showing extensive chromatolysis with displaced nuclei. The capil-

laries of the gray matter appear, in many instances, distended. A slight pial thickening is evident in the cervical region, but otherwise the meninges of the spinal cord are not altered.

The pathological changes in the pons are less intense than in the spinal cord. In the region of the pyramidal tract and the median fillet myeloclasts and a slight increase of the glia is perceptible. A few degenerated ganglion cells are likewise not infrequently encountered. In the red nucleus nerve cells showing fine granular disintegration of the chromatin usually associated with swelling of their cell bodies are occasionally seen.

In the medulla oblongata a diffuse but rather light grade degeneration is evident. In the olives and restiform bodies groups of degenerated nerve cells are seen in a few instances. The pathological alterations occurring in some of the ganglion cells are not unlike those described. Chromatolysis is the most frequent and characteristic change; more often of a peripheral than a central type. Sometimes disintegration of the Nissl substance is observed only in the protoplasmic processes.

Numerous sections from various parts of the brain were prepared by different methods of staining and carefully investigated. The changes in the frontal region are most discernible in the pyramidal and ganglionic layers. Some of the ganglion cells are well preserved, but the majority of them are altered. The nuclei are unusually large and filled with lightly stained granules. In proximity to the nuclei the cytoplasm presents a loose net-like structure, but around the periphery, and especially in the apical processes, contains numerous pale particles. Many of the pyramidal cells are swollen, and the alteration in their appearance is quite characteristic. The perinuclear cytoplasm has apparently in a number of instances undergone shrinkage, succeeding its initial distention. The dimly stained cell bodies and processes are entirely filled with pale ill defined granules, among which irregular masses of a more intensive color are frequently seen. The nuclei are voluminous and the nucleoli prominent. The nuclear membrane is sometimes scarcely distinguishable from the cytoplasm, but generally is more deeply tinged. In many of these cells, however, the nuclear membrane is exceedingly delicate, and the caryoplasm contains granules which so closely resemble those in the adjoining cytoplasm, that the limits of the nuclei can scarcely be determined. In addition to these alterations, a few cells which show a general shrinkage are encountered.

With Bielschowsky's method the axis cylinder processes are well preserved. Even in advanced nerve cell degeneration the neurofibrils are apparently but slightly affected. The reticular alteration occurring in some cells is quite distinctly demonstrated, but the neurofibrils in most appear intact.

An increase of the glia in this region is not pronounced, but progressive changes in much of the neuroglia are evident. The

alterations in the nucleus and protoplasmic structure are analogous to those observed in other areas. Gliogenic ameboid cells are sometimes seen, and a few glia cells showing regressive stages are visible.

The adventitial and endothelial cells of the cortical vessels, in some areas, contain yellowish-green pigment. These pigment granules are larger and more angular than those observed in the nerve cells. The adventitial spaces, especially of the veins, contain considerable lipoid substance.

In the precentral gyre, certain alterations are present, more obvious in some areas than in others. While a few of the Betz cells appear normal, a number of them are to a greater or less degree affected.

In many degenerative cells the nuclear membrane loses its usual affinity for the stain and is indistinct; the nucleolus stands out prominently and a pale ill-defined mass is discernible surrounding the nucleus. The structure of this substance simulates that of the altered nucleus causing the latter to appear enlarged. The cytoplasm of the cell is frequently more intensely tinged than normal and assumes an indefinite granular aspect rather than a reticular structure. The nucleoli in most cases exhibit alterations in contour and often contain small vacuoles, when extensive cellular disintegration is present. In certain parts of the cytoplasm, areas are seen which display peculiar changes. The stainable substance has undergone absorption and a granular net-like structure is perceptible. Considerable lipoid substance is to be found within a large proportion of the cells. These granules of fatty material have a pale lemon yellow tint by the Nissl method, are irregularly disposed and bear no definitely constant relation to any one part of the nerve cell or its processes, but vary in concentration and size.

With Herxheimer's method this substance presents a characteristic appearance, the individual minute particles staining a reddish color. They are mostly distributed in contiguity to the nucleus, especially its basal portion. In other instances, however, the fatty granules are seen around the sides of the nucleus, occupying a position between it and the darkly stained periphery of the cell.

Sections stained by the Bielschowsky method for the neurofibrils show but little alteration in these elements. The course of the neurofibrils is seen to be somewhat influenced, however, by the presence of the lipoid granules. A granular appearance of the neurofibrils is seen in some cells. Cellular disease of this type is more pronounced in some areas, but is encountered throughout this region in varying degrees of intensity.

Chronic disease of the nerve cells is also in evidence. This type of alteration is generally distinguished by an angular darkly stained nucleus and an enlargement of the nucleolus. The processes are thin, usually tortuous and visible for a considerable distance. The cytoplasm in some cells presents a peculiar granular appearance,

and in others contains numerous vacuoles. The changes in many of the cells are characterized by a more or less general shrinkage, and the cytoplasm and processes are filled with darkly stained coarse granules. The shrinkage is quite extensive in some instances so that the cell appears rather elongated. Combined types of ganglion cell degeneration sometimes occur.

With toluidin blue the neuroglia tissue is undoubtedly somewhat increased. Large glia cells having a distended lightly stained round nuclei are seen. Occasionally they are disposed in groups. Sometimes the nuclei of such cells are elongated and the cytoplasm at the poles deeply stained, so that they assume a rod-like aspect. Progressive neuroglia changes are constantly evident in the vicinity of degenerating ganglion cells, the intensity of the glia proliferations apparently corresponding to the degree of the cellular alterations. These glia cells are perceived in the various layers of the cortex, here and there as small aggregations surrounded by an ill-defined reticular protoplasmic structure, small round granules are usually found within the meshes of the reticulum, but quite frequently large irregularly shaped masses are present. In many instances the material within the cytoplasm stains a yellowish or yellowish-green color. In sections of the motor cortex stained by the Weigert-Pal method, it is obvious that neither the tangential layers of fibers nor the supraradiary and intraradiary plexuses are altered to any extent. The pathological changes in the cells of the temporal region correspond to those observed in the frontal gyri, but are somewhat less extensive.

The cellular alterations in the cornu ammonis are likewise interesting. The changes are chiefly confined to the stratum oriens and pyramidal cell layer. Certain more or less circumscribed areas are severely affected, whereas the cells in the adjacent tissue appear normal. Several types of nerve cell degeneration are observed. The nuclei in many cells are spindle or flask-shaped and intensely stained. Not infrequently an exoplasmic-like zone is discernible around the nuclei. The nucleoli are hardly visible within the dark homogeneous caryoplasm. When the degenerative process has reached an extreme stage, only slight or no traces whatever of normal elements are discernible in the cell bodies. The cytoplasm and somewhat tortuous protoplasmic processes present mostly a uniform aspect; but careful examination reveals the fact that their structure is composed of closely compressed palely tinged granules. The bases of the pyramids are angular and the basis process indistinct. Other cells within the affected regions show rarified areas within their cytoplasm, surrounded usually by a more deeply stained border, and which apparently represent beginning vacuolization of the cell body. Similar areas, but of a more irregular contour, are also seen in the processes. Ofttimes fine very dark granules are seen scattered through the protoplasm, probably products resulting from a metamorphosis of the unstainable substance.

On the other hand certain cells display a reticular-like degeneration.

Neuroglia proliferation is present where nerve cell diseases exist, chiefly of a progressive character. The *membrana glia superficialis* is not perceptibly thickened. In general only a moderate degree of proliferation is evident, but glia cells with large nuclei, distended metachromatically stained nucleoli, and a rather copious reticular cell body are visible. The nuclei of many cells are more darkly stained and normal in size, but around their periphery excessively tinged granules are distributed or scattered through the caryoplasm producing a stippled aspect. Sometimes the granules are closely massed together causing the nucleoplasm to appear homogeneous.

In the occipital cortex changes are perceptible in the majority of the nerve cells, but rather light grade in character. The cells in general are somewhat more deeply stained than normal, and the net-like structure of the cytoplasm less distinct than usual. Within the meshes faintly stained masses are discernible. A slight degree of alteration also obtained in the nuclei of many nerve cells, which is chiefly distinguished by a pale diffuse staining, but otherwise no distinct structural alteration is visible. The nuclei of the smaller cells, which normally have a greater affinity for the stain than those of the larger cells, show no deviation from the normal. Besides the above described apparently inconsiderable changes, a number of cells are encountered displaying transitional stages of degeneration, until nothing remains but an indistinct profile of the cell body, which occasionally contains a few more or less sharply defined vacuoles. In the upper layers of the cortex some cells are severely altered, and acute degenerative changes are not infrequently met with.

The neuroglia tissue throughout the cortex in this region is but little altered. Glia cells with darkly stained or pycnotic nuclei are seldom in evidence. Likewise relatively few cells with large pale nuclei are seen. The nuclei of such cells often show a metachromatic tinctorial reaction and centralization of the chromatin. Proliferating glia is occasionally encountered. In other instances regressive changes are perceived, as indicated by the small angular-shaped homogeneous appearance of the nuclei and degeneration of the cell body. Examination of the basal ganglia shows no pronounced degenerative changes in the neurons of this region.

The white matter of the hemispheres was investigated by the Nissl, Marchi, Mann-Alzheimer, Mallory and other methods. In the subcortical white matter of the motor and frontal regions disintegrating fibers are occasionally seen. With Marchi's method these degenerating fibers are often perceived as rows of fat granules, or in cross sections sometimes present a radiative appearance. In counterstained sections, it is readily demonstrated that

these masses are inclusions within the protoplasmic structure of metamorphosing glia cells. The Mallory and Mann-Alzheimer methods show the complicated cytoplasmic structure of these cells, and a fine rather uniform meshwork can be traced through their protoplasmic cell bodies. The net-like cytoplasm is frequently copious and extending into the surrounding area, gradually merges with similar elements, so that its limits cannot be defined. Lying near the nucleus of some cells, large round or oval-shaped vacuoles are apparent. Such cells often possess long thick processes, which penetrate the neighboring tissue, and undoubtedly play an important rôle as supporting structures. Glia cells with small dark nuclei and delicate cytoplasm are also in evidence. A similar microscopic picture is discernible with the Nissl stain.

Cerebellum.—No change of any significance could be detected, only a few of the Purkinje cells are altered, and but slight increase of the neuroglia elements is appreciable.

GENERAL PATHOLOGICAL SUMMARY

The brain appeared somewhat edematous, but its convolitional pattern was normal. There was no disease of the membranes, other than slight opacity of the pia-arachnoid in the interpenduncular space. The cerebral vessels showed nothing abnormal, although some of the veins were distended.

Microscopically pathological alterations were discernible in both the cortex and white matter of the hemispheres. The changes in the cortex from a cyto-architectonic point of view were most intense in the precentral and frontal regions. A swelling of the ganglion cells was the most frequent alteration observed, but various types of cell disease were encountered, invariably associated with hyperpigmentation.

An increase in the neuroglia tissue was not pronounced, but progressive changes in the glia were in evidence, characterized in some instances by proliferation and enlargement of the nucleus, a greater intensity in the staining reaction of the cytoplasm with a deposition of basophilic substance and the formation of small aggregations surrounded by an ill-defined reticular protoplasmic structure; and in other instances by the formation of fibers. The intensity of the glia proliferation apparently corresponded to the degree of cellular alterations.

In reference to the medullated fibers of the cortex, neither the tangential fibers nor the supraradiary and intraradiary plexuses were altered to any extent.

A careful investigation of the white matter revealed subcortical areas of fiber degeneration principally confined to the motor and frontal regions.

Sections from the cervical and dorsal cord showed definite degeneration of the posterior columns and a light-grade diffuse alter-

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ation was perceptible in the lateral columns. The degeneration was most extensive in the cervical region affecting here the whole of the posterior columns except a narrow rim bordering the posterior commissure and horns, and gradually becoming less pronounced towards the lower cord. In the lumbar region a small flask-shaped area of degeneration was present near the center of each posterior column. The degeneration was of an irregular character throughout the entire length of the cord, and presented a somewhat patchy distribution, often varying at different levels. The posterior roots were not affected. The changes in the lateral columns were not as well defined as those involving the posterior tracts, but were likewise most evident in the cervico-dorsal region. The degeneration was less regular and more patchy in distribution and while not extending over so great a length of the cord, was, however, discernible to a slight degree in the lumbar region. Neither the ventral spino-cerebellar nor the anterior pyramidal tract was altered. In the degenerated areas a disintegration of the medullary sheath and axones of the nerve fibers occurred, associated with an increase of the neuroglia tissue. No hemorrhages were apparent. As regards the gray matter, most of the cells in the anterior horns were unaltered, but in some of them evidences of degeneration were perceptible. The cells of the posterior appeared somewhat more intensely altered. The capillaries were in many instances distended, but the vessels of the cord in general showed no special abnormalities.

The pathological study of this case is of special interest and value, since anatomical alterations not only occurred in the spinal cord, but were demonstrable in the brain.

The changes affecting the nerve cells in the brain and spinal cord simulated those alterations of the neuronic elements which are encountered in states of chronic and subacute toxemia, and obviously resulted from the action of the same toxic agent responsible for the degenerative changes in the white matter.

Only a few cases of this affection are recorded in the literature which exhibited cerebral lesions. Biruljas reported a case in which an examination of the brain showed small hemorrhages and degenerative changes in the ganglion cells. Similar alterations have been found by Ransohoff. More recently Lube has observed foci in the cerebrum and cerebellum analogous to the areas found in the spinal cord.

The degeneration which occurs in the spinal cord assumes the character of what has been termed pseudocombined sclerosis and has been described by most authors as a patchy, or irregular de-

generation and sclerosis confined to the posterior columns, or not infrequently combined with a similar process in the lateral tracts.

A number of views have been advanced regarding the etiological and pathological factors in this affection, and the spinal cord changes have been attributed to several different pathological processes.

Some of the earlier writers (Minnich, von Noordan, Teichmüller) were of the opinion that the sclerosis resulted from a coalescence of multiple small hemorrhages. This hypothesis does not, however, appear to be well founded, as many subsequent investigators have failed to detect the presence of hemorrhages, nor could any evidence of hemorrhagic extravasations be discovered in the case considered in this paper. It is also apparent that when hemorrhages have been observed they were too small, few in number and irregularly scattered to be regarded as accountable for the production of degenerations, affecting in many instances a large part of the spinal cord. On the other hand, should large hemorrhages occur in the cord which interrupted parts of similar tracts on either side, there would be evidences of ascending degeneration of certain tracts above such lesions with their partial escape at least below them, and of descending degenerations of other tracts below the hemorrhages, with partial or complete escape of the same tracts above them. Hemorrhages of this character have not been described, and the distribution of the degenerations in the spinal cord contraindicates such a possibility. It should be remembered that in the majority of the cases, the degeneration both in the posterior and lateral columns is most marked in the cervical and upper thoracic regions of the cord and decreases in extent and intensity towards the caudal extremity.

A consideration of vascular alterations as a possible factor in the production of the degeneration seems appropriate, since various degrees and types of vascular disease have been described in severe anemia.

Thickening of the walls of blood vessels, producing secondary changes in the nerve tissue, has been suggested (Nonne, Redlich, Minnich). Although areas of degeneration surrounding vessels have been found, such areas have as frequently occurred independent of the blood vessels, and there seems to be no justification for the assumption that the affection has a vascular origin, in the

sense that the vessel walls are primarily diseased and the nerve elements secondarily involved.

Thickening of the vessels would unquestionably cause a diminished permeability of their walls, and in such instances disturb the nutrition of the areas supplied by the vessels and a consequent impoverishment of the nerve tissue, but that this should be considered *per se* as the prime etiological factor seems most improbable.

Thrombosis of the spinal cord vessels has been described by some writers (Dana, Henneberg, Petren), and it is interesting to note that when transverse softenings occur in the spinal cord, apparently due to thrombotic disturbance, they appear to have a predilection for regions similar to those most extensively involved in pernicious anemia, which might lead one to assume that the cervical upper thoracic regions of the cord are less efficiently supplied with blood than other parts.

Anatomical data, however, do not seem to support the assumption as most of the evidence at hand indicates that the lower part of the dorsal as well as the lumbar and sacral regions of the cord, are not as well supplied with blood not only owing to the fact that this portion of the spinal cord is most distant from the origin of the anterior and posterior spinal arteries, which arise from the vertebral arteries within the cranium, but because the arteries which pass to the spinal cord via the nerve roots have a longer and more vertical course to this part of the cord.

The seemingly plausible vascular theory of Marie, which attributes the simultaneous affection of tracts in the dorsal half of the spinal cord to a lesion of the posterior spinal artery, is also untenable, as researches regarding the vascular topography of the spinal cord preclude this presumption. Jakob and Moxter, in a series of five cases found an intense intima proliferation, in some instances obliterating the lumen of the vessel. This proliferation not only involved the vessels in the degenerated regions, but occurred through the entire spinal cord, and doubtless represented an independent process.

If the vascular disease were invariably confined to the areas of degeneration, a relationship between the two processes might be indicated.

It has been proposed that the alterations in the coats of the vessels, described by some writers, were the result of the same

toxic process which had produced disintegrative changes in the nerve fibers. This hypothesis which apparently premises that the toxin permeates the vessel walls without injury to their structure, causes a degeneration of the surrounding tissue, and a subsequent alteration in the coats of the vessels, offers no adequate explanation, as it seems unlikely that a toxin capable of effecting in a comparatively brief period of time, extensive morbid changes in the nerve tissue, would fail to occasion initial vascular lesion. Nonne has referred to the possibility that a perivascular lymph stasis, demonstrated by distention of the perivascular spaces, may through pressure and irritation of the vessel walls induce proliferative processes terminating in sclerosis. Dilatation of the perivascular spaces can hardly be considered as indicative of lymph stasis, and it is most probable that in such instances the distention was the result of post-mortem changes or the action of fixatives.

In the more recent literature, however, a number of cases are recorded in which the vascular alterations are inconsiderable in character, the prevailing type consisting of a slight adventitial swelling and with a normal condition of the media and intima. Proliferative changes in the intima have been rarely described, and but few authors have observed any evidence of inflammatory processes affecting the vessel walls.

The vascular alterations in our case were also of a trifling nature, and could not be regarded as bearing any etiological significance to the degeneration in the spinal cord.

The slight distention of the lymphatic spaces of the vessels, as well as the associated adventitial changes, while not constantly present, can be readily comprehended when the histological features of the reactive process in general are analyzed. The morbid changes in the white matter of the cord were characterized by degeneration of the nerve fibers, with a compensatory glial metamorphosis tending to terminate in sclerosis. This process was distinguished by several stages and numerous transitional types of gliogenous phagocytic cells were discernible. Many of these cells were laden with products of disintegration, to be conveyed from the ectodermal tissue to the vascular system; and it was observed that where these cells accumulated about the blood vessels, the lymphatic spaces were filled with the detritus, and in consequence enlarged. The cytoplasm of the adventitial cells contained similar material. Hence it is obvious that the distention of the vas-

cular lymph spaces and the adventitial alterations, represent but the activity of the vessels as participants in the disposition of waste products.

Myelitis was considered as the fundamental etiological process by Nonne and others. A number of investigators, however, have failed to detect any changes in the spinal cord characteristic of what might be considered as a myelitic reaction; and, the reasons advanced in discussing why the degenerations were not the result of hemorrhages, are relevant in the case of myelitis.

The alterations in the white matter have been regarded as consequent to a primary disease of the gray matter (Rothmann, Boedecker, and Juliusberger). There is considerable evidence against the acceptance of this inference. It seems more likely that the elements of the gray matter are affected simultaneously with, or subsequently to those of the white substance, through the activity of the same toxin.

Combined tract degeneration has also been observed in a number of toxic, cachectic or anemic conditions. Changes in the spinal cord have been reported in leukemia (Bloch and Hirschfield, Nonne, Müller); chronic alcoholism (Vierodt, Redlich, Braun); arsenic poisoning (Dana); diabetes (Williamson); lead poisoning (v. Monakow); pellagra (Marie); ergot poisoning (Tuczek); etc. Such observations lend support to the theory that a toxic agent is responsible for the tissue changes. To explain why in many cases some parts are affected and others escape, it seems most reasonable to assume that all parts of the nervous system are not equally vulnerable to the deleterious effect of the same toxin.

The conclusions to be deducted from this study, are that cerebral alterations can be demonstrated in progressive pernicious anemia, and that in cases with psychical phenomena, changes in the cortical neurones occur similar to those found in the psychosis of toxemic origin.

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Society Proceedings

NEW YORK NEUROLOGICAL SOCIETY

OCTOBER 6, 1914

The President, DR. SMITH ELY JELLIFFE, in the Chair

DOUBLE CERVICAL RIB NERVE SYMPTOMS: SURGICAL REMOVAL

By S. P. Goodhart, M.D., and Alfred S. Taylor, M.D.

The patient was a woman, 30 years old, whose history, both family and personal, was entirely negative from a neuropathological point of view. Symptoms referable to the cervical ribs dated apparently from the patient's eighth year, when she began to suffer from pains in the left shoulder radiating down the arm, and the entire left upper extremity would become paroxysmally numb. There was also a gradual development of an indefinite sense of discomfort about the left shoulder and a protrusion of the left shoulder blade, which, when pressed upon, gave rise to varying degrees of pain and numbness extending through the arm. These symptoms persisted, and in the course of a few years there was added a certain inability to properly use the left index finger, noticed particularly in playing the piano, and gradually a lack of dexterity of the left hand. During her 18th year she noticed that certain movements of the arm, for example, arranging her clothing necessitating placing the arm behind her, caused flexor spasm of the muscles of the forearm and hand. At this time there was no weakness nor wasting of the muscles.

When the patient was 23 years old she observed weakness in the grasp and in the finer movements of the left hand, followed by progressive atrophy of the small hand muscles, both the thenar and hypothenar surfaces being involved. Soon afterwards a coldness of the left upper extremity, particularly from the elbow to the finger tips, was at times subjectively felt and could be objectively demonstrated.

The patient came to Dr. Goodhart in 1904 with the diagnosis of progressive muscular atrophy. At that time his examination revealed areas of hypesthesia and hyperalgesia irregularly distributed over the left forearm and hand. These areas varied and finally disappeared, leaving as the only sensory disturbance an area of hypesthesia for all forms of sensation in that part of the forearm and hand corresponding to the inner cord of the brachial plexus, particularly the ulnar distribution. At this time the left hand presented an appearance of the typical *main en griffe*, and some atrophy of the pectoral muscles was noted. Further physical examination revealed osseous tumors in each supraclavicular space, which were recognized as cervical ribs. The one on the left side was found two inches outside of the sternal insertion of the sterno-cleido-mastoid muscle, extending vertically upward two inches from the upper clavicular border.

On the right side a far smaller eminence was found in about the same position. The x-rays disclosed scoliosis extending from the 6th cervical to the 5th dorsal, with the convexity to the left side, and with a compensatory convexity below. Bilateral cervical ribs were seen attached to the bodies of the 7th cervical vertebra, articulating at their distal ends with a facet from the upper border of the first dorsal ribs. On the left side the supernumerary rib measured about one and three quarters inches; the right one about two inches in length.

About 1907 the patient began to observe a weakness in the right index finger and a certain lack of dexterity in executing finer movements with the right hand: slight occasional numbness of the right thumb was also complained of. Thus, involvement of the brachial plexus on the right side developed. Within the next few months numbness of the entire right leg and pains in the calf muscles were experienced by the patient, and Dr. Goodhart was able to detect at times a slight but manifest weakness in the flexors of the toes of the right foot, but there were no secondary nor electrical changes.

In May, 1908, the patient submitted to an operation for the removal of both false ribs, which was performed by Dr. Alfred S. Taylor. Upon exposure, it was seen that the subclavian artery crossed the first rib anteriorly and below the distal end of the cervical rib, the roots of the plexus lying over the false rib, as had previously been determined by palpation. The 8th cervical and 1st dorsal roots were undergoing the greatest tension and pressure. This coincided with the symptoms. The relation of the structures was about the same on both sides, and the upper roots were so situated as to be but little compressed.

Immediately following the operation there was paralysis of nearly all the muscles and the sensory surfaces supplied by the plexus on both sides. In the course of a week, motion and sensation began to return, and the improvement, though slow, was constant. About one year after the operation there was still considerable atrophy and very slight sensory changes over the inner surface of the forearm.

At the present time, about six years after the operation, there is marked improvement in the muscle supply.

Dr. Richard B. Kruna suggested that the persisting deformity of the hand and arm might be improved by a systematic course of exercises of the involved muscles and nerve tracts.

Dr. Charles A. Elsberg, in reply to an inquiry as to whether he thought this patient might be benefited by further operative interference, said her symptoms were incomplete and there was no reason to think there had been sufficient injury to the branches of the plexus to justify an operation which would necessitate a complete division of the nerve cords and subsequent suture. Through reëducation and systematic exercises, as suggested by Dr. Kruna, we might get other nerves to take up the function of the impaired muscles.

A CASE OF SPINAL HEMATOMYELIA OF THE HEMIPLEGIC TYPE

By Frederick Tilney, M.D., and C. L. Nichols, M.D.

The patient was a printer, 28 years old, who was admitted to the Coney Island Hospital on June 18, 1914, with the history that shortly

before admission, while diving in shallow water, his foot slipped and he fell into the water almost perpendicularly. He did not recall whether he struck the sandy bottom on his head, neck, or back, but thought that it was on the vertex, posteriorly. He had to be dragged from the water and was found to be suffering from a complete paralysis of the right upper and lower extremities, and an awkwardness in the use of the limbs on the opposite side. There had been no loss of consciousness and he was perfectly oriented. His head was numb and the whole body felt bruised and sore. The pain was most severe over the right shoulder, extending down the arm and forearm. There were no spasmodic movements in this area.

When the patient was examined several hours after the injury he still complained of the sensation of having been bruised, especially over the right clavicle and upper chest. He was exceedingly sensitive from a sharply defined point at the sterno-clavicular articulation outward to nearly the tip of the shoulder. The right pupil was smaller than the left: both were symmetrical and responded to light and accommodation. The right side of the neck was moved with great reluctance, and posteriorly there was a certain amount of rigidity. The right side of the chest was less mobile than the left, and the breathing sounds over the former were diminished. The heart's action was slow and regular and the sounds of good character. The pulse was of fair quality and volume. The abdomen showed no abnormalities. No reflexes could be elicited on the right side, and sensation was absent, with the exception of variable areas of response to pin prick. The field of vision was undisturbed. The fundus showed slight congestion of the nerve; the margin was not well defined and the vessels were slightly engorged, evidencing some pressure. The hearing was affected on both sides.

On examination on June 25, one week later, the anisocoria was still more apparent, the left pupil being larger than normal and reacting promptly, while the right was smaller and sluggish. The patient was placed on a water bed for two weeks, during which time the soreness gradually cleared up and the paralysis improved. He was then permitted to sit up, supported by pillows, and to use a mattress bed. Two weeks later he was allowed to sit in a chair and ten days after that he was able to walk. He remained in the hospital a month and a half.

On the first day after his discharge he had an agonizing pain on the right side of the head over an area extending from the forehead along the vertex in the median line to the occiput. This persisted for two hours, gradually increasing in severity and then diminishing slowly after the application of cold compresses. A similar attack, less severe, followed a week later.

After the patient had been seated for any length of time he became stiff in the right hip, and on arising had a dull pain extending from the lower part of the spine forward into the right groin. This symptom had been noticed irregularly, averaging once a day, since he had been permitted to sit erect in a chair at the hospital. He also complained of a similar pain extending from the cervical spine to the right shoulder. There were no urinary symptoms. The bowels had moved regularly ever since the accident.

Examination at the present time showed a flaccid paralysis of the right upper extremity and a spastic paralysis of the right lower extremity. The tendon reflexes on this side were greater than those on the left,

while the abdominal, epigastric and cremasteric reflexes were absent. The latter were present on the left side. The Babinski was positive on the right side, and there was permanent clonus in the ankle. A peculiar circumstance in connection with the case was that in eliciting the triceps reflex on the right side, percussion on either the tendon or the muscle produced flexion of the forearm instead of extension. The radiograph showed a fracture of the sixth cervical vertebra.

A CASE OF SPINAL HEMATOMYELIA: BROWN-SÉQUARD PARALYSIS

By Frederick Tilney, M.D., and C. L. Nichols, M.D.

The patient was a boy of twenty, an electrician, who was admitted to the King's County Hospital on November 10, 1913, with the history that just previous to admission, while climbing a tree, the branch broke, and he fell a distance of about ten feet, first striking upon his buttocks on a branch below, and then falling to the ground on his chest. He lay flat on his face, without losing consciousness, the fingers of both hands contracted spasmodically. None of the extremities could be moved voluntarily, and he felt as though a current of electricity had passed through him, leaving the body charged from the shoulders down. The right lower extremity seemed to contain a tremendous pressure, and a tingling sensation passed up the left leg, across the pelvis and down the other limb. The direction did not change.

Physical examination showed an apparently healthy and well-developed adult, rather nervous, with a slight tremor of the hands. The pupils were equal, and reacted naturally. The pulse was regular and of good volume. The heart sounds were normal. The respiration was chiefly abdominal, the chest moving very little. The arms could be moved, though very slowly and with great difficulty. The fingers were held in a position of partial flexion, with no power to either flex or extend them. The knee jerk was absent on the right side; present on the left. The Babinski could not be elicited. The toes of the right foot were red; those of the other pale, and the surface temperature of the left lower extremity was diminished. There was an absence of the cremasteric reflex.

There were marked sensory changes. There was tactile anesthesia in both lower extremities and in the trunk up to the second interspace. Pain sense was absent in the left leg and as far up as the third rib, but present over a corresponding area on the opposite half of the body, where it was apparently hyperacute. The temperature discrimination was entirely absent on the left side to a level a little below the clavicle, but was present on the right side, although the response to heat was not as good as to cold. There were sticking pains in the right leg, and the patient felt as though there was a strap around it and another around the shoulders, each being tightened in an opposite direction. There were fibrillary tremors on the left side. Priapism was present during the examination. Catheterization showed no evidence of hematuria.

One month later the patient had slight power of flexion of the left fingers. Sensation was unchanged. The pupils reacted promptly, but were unequal. The patellar reflex on the right side was exaggerated. One week later there was an involuntary urination. Up to that time catheterization had been necessary, but from that time on the control of the bladder was normal.

The lesion in this case was located in the upper cervical region. The radiographs demonstrated no fracture nor dislocation.

A CASE OF SPINAL HEMATOMYELIA OF THE PARAPLEGIC TYPE

By Frederick Tilney, M.D., and C. L. Nichols, M.D.

The patient was a Russian tailor, 34 years old, who came to the Vanderbilt Clinic on September 16, 1914, complaining of inability to walk properly, with pain in the back when seated, swelling and coldness of the feet and lack of control of the bladder. The family as well as his previous history was negative.

Nine months ago, in an attempt to escape from a burning building, the patient either fell or jumped from a fire escape, a distance of two stories. He was picked up in an unconscious state and removed to the Gouverneur Hospital. He remained unconscious for two hours—perhaps longer—and on recovering his senses he was unable to move either lower extremity and had retention of urine. He remained in the hospital for six weeks, with slight improvement. He was then taken to the Beth Israel Hospital, where he remained three months and where some operation was done. Following this his condition improved and he regained some power to move the legs. He was now able to walk, with some difficulty.

Examination at the present time showed normal cranial nerves and normal reflexes, with the exception of those at the patella and ankle, which were exaggerated. The station was unsteady and the gait spastic. Motor power in both extremities was defective, with some atrophy of the muscles. There was a fine tremor of the hands and tongue. There was an area of anesthesia over the left gluteal region.

Dr. Frederick Tilney, who had seen the first two cases of Dr. Nichols in their early stages, said that in both instances he had concluded that the condition was due to hemorrhage into the cord. In the second case, that showing the Brown-Séquard syndrome, the question of an operation was considered by Dr. Arthur C. Brush who advised against it. The improvement in this case was so marked during the first three months after the injury that further delay was advised, and the patient was now able to walk, whereas when he was brought into the hospital he was completely paralyzed. In the first case, the patient with hemiplegia following a diving injury, the radiograph showed a fracture of the sixth cervical vertebra, but this was of such a character that the symptoms seemed to be due to hematomyelia rather than to a cerebral injury or a crushing of the cord. In this case an expectant plan of treatment was also followed and the patient had improved steadily.

One of the reasons why these cases were shown here tonight, Dr. Tilney said, was to get an expression of opinion from the members as to whether early operative interference was advisable in dealing with injuries of this character.

Dr. William M. Leszynsky said that in cases of traumatic lesions of the cord, of which he had seen a large number in hospital practice, the question of operation usually came up for discussion, and in answering it he had been largely guided by the clinical manifestations. If either by

palpation or the x-ray it could be shown that we were dealing with a fracture dislocation producing crushing of the cord, as evidenced by complete motor and sensory paralysis and abolition of reflexes below the lesion, an operation was inadvisable. When blood is found in the cerebrospinal fluid, the hemorrhage is usually subdural. When the cerebrospinal fluid is clear, as in hematomyelia, operation is not indicated.

In many of these cases, Dr. Leszynsky said, the early residual symptoms often became permanent, their severity depending upon the amount of damage done to the cord. It was impossible to give a positive prognosis as to the ultimate outcome.

Dr. Benjamin Rosenbluth said that the first case presented by Dr. Nichols he had seen at the Beth Israel Hospital, the man had been previously treated at the Gouverneur Hospital and was brought in with a complete flaccid paralysis of the lower extremities, anesthesia, loss of reflexes and localized pain over the spine. He was put in plaster but this proved so painful that it had to be taken off. Several radiographs were taken with no definite findings. A localized injury involving the body of the vertebra and transverse processes and possibly the cauda equina was suspected, and when the spinal canal was opened over the region of the cauda the nerves were found to be pushed about in all directions and there was a large collection of cerebrospinal fluid. This pressure was relieved and the man made a good recovery so far as motility was concerned, but he still had an area of anesthesia over the thighs. Dr. Rosenbluth said that was the fourth case of injury of the cauda equina that had come under his observation, and the result of the operation was the most encouraging he had seen. The symptoms were not hemiplegic, and his result showed a larger sensory disturbance than reported.

Dr. J. F. Terriberry, in reply to Dr. Tilney, said that in cases where one could be fairly certain that there was a hemorrhage within the cord, little could be expected from operative interference. This could perhaps be determined by the x-rays together with the clinical history. In the cases shown by Drs. Tilney and Nichols the history clearly indicated a hemorrhage into the cord itself, and under those conditions no benefit could be derived from an operation. The spinal cord is a very sensitive organ, as we all know, and any injury to it might be aggravated by operative interference. These cases, as a rule, did much better under proper general care, with subsequent reëducation of the lost muscular function if the residual symptoms demanded it. The fact should be borne in mind that this loss of function was sometimes attributable to a stiffening of the joints of the hands and fingers, which often did much to retard the reparative process, and it was only with care and perseverance that one could get the most out of these cases.

A CASE FOR DIAGNOSIS

By A. Skversky, M.D., and Smith Ely Jelliffe, M.D.

The patient was a child, seven and a half years old, of normal birth, who sat, talked and walked at the proper ages, and appeared to develop normally. She had measles at the age of three years, with an uneventful recovery. Six months later she was said to have had a high fever one night, but no residuals followed. Five weeks after this, without any

premonitory symptoms and without convulsions, she had a sudden loss of power on the left side of the body involving both extremities and the face, without speech disorder. Two weeks later, according to the mother's description, she had epileptiform seizures, apparently Jacksonian, for they began in the left lower extremity, ascending, involving the upper extremity and face, and terminating in a generalized convulsion. This occurred only once, and following this her hemiplegia was said to have disappeared and she was perfectly well for three months, when she began to have irregular involuntary movements of the extremities on the left side, accompanied by some difficulty in walking. This had been becoming slowly but progressively worse.

For the past year she had headaches at irregular intervals, but excruciating, and lately they had become more continuous and dull, usually localized in the right frontal region. She had also complained somewhat of dizziness. There was occasional nausea, but no history of vomiting. There had been no difficulty with vision or hearing. The mother claimed that the child's speech had always been somewhat drawling and monotonous. She was rather bright, but had received no schooling and became emotional by slight stimulation. For the past month she had had some difficulty with bladder control, with occasional incontinence of urine.

When the patient was brought to the Post-Graduate Hospital, about a month ago, her chief complaint was difficulty in walking. Examination showed a well developed and nourished child. Her mental level, according to the Binet-Simon rule, was only a little less than a year below her age, and she was therefore not feeble-minded. There was marked ataxia, with mild left hemiparesis. The more important positive neurological findings were as follows: The head was usually inclined towards the right, and the face tilted towards the left. Vision was normal, but the fundi presented double optic neuritis, more marked on the right side. At rest, there was slight ptosis on the right side, and also weakness of the right internal rectus. No nystagmus. The pupillary reactions were fairly normal. The fifth nerve was intact: there was slight facial palsy on the left side when crying. Hearing was normal. The speech was drawling in character: swallowing normal.

The left upper extremity showed no atrophy nor hypertrophy, but marked hypotonicity and slightly decreased muscular power. The reflexes were present. There was marked incoördination, often resembling a very coarse intention tremor. There was marked adiadochokinesis: the stereognostic sense was intact: there was some dissociation of motion and position and also temperature sense; there was general hyperesthesia, apparently emotional, with contradictory replies and lack of proper coöperation. The right upper extremity showed a slight incoördination on volition. The abdominal and epigastric reflexes were present, with marked asynergia of the trunk.

The left lower extremity presented a similar picture to the upper, but more pronounced. There was hypotonicity and decreased muscular power. The patellar and Achilles reflexes were present, but not exaggerated: no Babinski nor other extensor responses. On a few occasions, a double exhaustible ankle clonus was obtained. There was marked impairment of synergistic sense, as well as definite ataxia, not increased on closure of the eyes. The superficial reflexes and sensory symptoms were similar to those in the upper extremity, occasionally dissociated, but contradictory.

The authors stated it was evident from the symptomatology present in

this case that one of the factors they were dealing with was that of increased intracranial pressure, as shown by double optic neuritis, headache and vertigo, and, more recently, somnolence. Syphilis was ruled out by negative blood and spinal fluid examinations and by the family history. There then remained the possibility of neoplasm, cysts or internal hydrocephalus.

If one assumed that one were dealing with a neoplasm, which was quite likely, because the process had been a slowly progressive one, the problem of localization in this case became a rather difficult and uncertain one. There was a good deal of evidence suggestive of chronic cerebellar disease, as shown by the vertigo, the markedly ataxic gait, asynergia on voluntary movement and mild hemiparesis, together with a definite adiadochokinesis and unaltered plantar and inconstant deep reflexes. There was, however, no nystagmus.

As to cerebello-pontine angle involvement, there was of course a mild facial palsy, but no disturbance of hearing, and it seemed rather unlikely that an extracerebellar lesion in this region, which was usually of auditory nerve origin, should not produce some disturbance of hearing.

In view of the very early appearance of Jacksonian convulsions—two weeks following the initial hemiplegia—it was difficult to rule out a motor cortex disturbance, and with the few definite localizing signs, it was suggestive of a supratentorial process, probably subcortical. It was therefore quite likely that a progressive lesion in the post-frontal region would cause contiguous pressure phenomena, and in addition interfere with the fronto-cerebellar tracts, giving the signs simulating cerebellar disease. The optic neuritis, generally late in onset, would fit in here.

The convulsions might be explained by a lesion in the vermis, which usually gave rise to so-called cerebellar fits, and therefore completed the picture of a distinct cerebellar affair. Similarly, the post-parietal area so intimately associated with cerebellar functions might easily account for nearby pressure signs, but the usual sensory disturbances were lacking: also the stereognostic and visual functions were intact.

The child had often displayed choreo-athetoid movements on the affected side, increased on exertion: this, with her increased emotional reaction to external stimuli, would suggest a thalamic lesion, but the sensory symptoms, especially the subjective paroxysmal pains, heat and cold, and the hemianesthesia were absent. If only the posterior end of the thalamus be involved, superior cerebellar peduncle disturbance would answer well.

As to the nature of the lesion, if we realized that the disease was probably of three and a half years' duration in a child seven and a half years old, it was difficult to ascribe it to the characteristic ones, namely, sarcoma, glioma, tuberculosis, cyst, etc. The optic neuritis was apparently of recent onset, because the vision was fairly intact. Tuberculoma was rendered improbable by the absence of signs elsewhere, while sarcoma or glioma should have done more damage by this time unless there be a recession, with cyst formation. Internal hydrocephalus could be fairly ruled out by the absence of sufficient mental impairment, lack of motor tract disturbance and gross appearance of the head. The most likely lesion would be the relatively benign endothelioma.

Dr. Leszynsky said the history of the case seemed to indicate an encephalitic process to begin with. What the condition was now he did not know, but the symptoms pointed to a cerebellar lesion.

Dr. Tilney said that from the confusing symptoms, it was difficult to decide upon any particular localization. Still, the ataxia pointed quite definitely to a cerebellar lesion, and as we knew that that part of the brain is so frequently affected in children, he would be inclined to ascribe the symptoms to a lesion involving the dentate nucleus on the left side.

Dr. S. P. Goodhart said that in the absence of nystagmus, he was not inclined to accept the assumption of Dr. Tilney, and he thought that a post-frontal right-sided lesion could not be positively ruled out. In a hypophysial tumor it was not uncommon to find the field of vision unaffected.

Dr. Rosenbluth, who had previously seen this patient at the Beth Israel Hospital, said that while the history he obtained from the mother at that time was not very clear, it pointed to a meningitis in infancy, and the convulsions seemed to be post-encephalitic, apparently of the motor cortex type. The eyes at that time gave negative findings. He regarded the case as one of left cerebral hemiplegia, with post-frontal sensory disturbances.

Dr. Terriberry thought the symptoms pointed to a preceding inflammatory process, and gradually progressing from an irritative into a compressive condition. The symptoms seem to indicate that the trouble, at present, is confined to the sensory apparatus. It is impossible to trace these symptoms to a small focus. We probably have to deal here with what was at first a rather widely distributed irritation which had now become restricted to a basal region fairly well forward. He was not inclined to believe that we had to deal with a neoplasm. Possibly there was an internal hydrocephalus as a part of the picture.

Dr. Leszynsky thought the authors had scarcely been justified in ruling out an internal hydrocephalus, while a subtentorial process, which they favored, would probably have given rise to more pronounced papillitis. In all probability, the speaker said, the child had an encephalitic process which produced the original symptoms, namely, the convulsions and subsequent paralysis.

Dr. Skversky said he did not think the fever bore any relation to the disease process.

A CASE FOR DIAGNOSIS [SPECIFIC LESION INVOLVING THE THALAMUS?]

By Smith Ely Jelliffe, M.D.

The patient was a married woman, 32 years old, the mother of three children, aged, respectively, nine, seven and two and a half years. The patient's history dated back to March, 1913, when she began to suffer from pain in the left side of the body and the left arm and hand, with numbness, and pain in the neck on the same side. These pains were rather severe, and of a sharp, shooting character, and have been more or less constant from that time on. Following this, there was a slowly progressive numbness and tingling, with a sense of insecurity on the left side of the body and face, and she noticed that she began to drop objects that she held in her left hand. There was also a slight irregular tremor of the left hand when she began to use it.

Examination showed a definite impairment of epicritic sensibility in

the hands and feet and a loss of stereognostic sense. The reflexes were increased on both sides, but not more marked on one side than the other. There was no Babinski; no ankle clonus. There was a definite loss of ability to pronate and supinate the left side without impairment of muscular power (adiadochokinesia). There was no interlacing of the color fields, but the eye-charts indicated the onset of a gradual bitemporal field contraction for all colors, premonitory to a bitemporal hemianopsia. The Wassermann was originally negative, but under a provocative neosalvarsan injection it became double plus; subsequently it again was negative. The x-ray showed no changes in the sella turcica. The patient complained of a crackling noise in the left ear.

During the earlier stage of this patient's illness, Dr. Jelliffe said, there had been a great deal of emotional hyperactivity with great asthenia which had led to a previous diagnosis of hysteria.

Dr. Louis Casamajor said the sensory and motor symptoms in this case led him to think of a thalamic lesion. He could recall instances where for a long time such a lesion gave rise to symptoms which were regarded as functional, before its organic character was recognized. This assumption of a thalamic lesion, however, was not in accord with a bitemporal hemianopsia, as it was difficult to conceive how a single lesion could reach from the posterior part of the chiasm upwards and not involve the pyramidal tracts.

Dr. Terriberry said the eye chart shown by Dr. Jelliffe did not indicate a bitemporal hemianopsia: it only showed a concentric contraction of the visual fields. The symptoms in this case reminded him of one that recently came under his observation in which he made a diagnosis of hysteria, which subsequent events proved to be correct.

Dr. Tilney said that with the bitemporal hemianopsia eliminated, he thought the symptoms pointed to a thalamic lesion.

Dr. Walter Timme thought the symptoms indicated a thalamic lesion. It had lately been reported that the subthalamic lesions had some effect on bladder control, and this fact would suffice to bring the lesion producing the syndrome shown by the patient in the hypothalamus.

Dr. Rosenbluth suggested a multiple sclerosis with unusual symptoms, most likely a luetic process. In such a case we might have to deal with a thalamic lesion, as cases he had seen with multiple sclerosis symptoms with sudden onset, had shown thalamic lesions postmortem.

Dr. Goodhart thought the indications were in favor of a vascular lesion of specific origin.

Dr. Jelliffe said the suggestion made by Dr. Rosenbluth was interesting in view of the fact that the Wassermann test was positive in some cases of multiple sclerosis.

Personally, the speaker said, he had no doubt that we were dealing here with an organic lesion. These thalamic cases were not infrequently looked upon as functional because of the fact that the symptoms were purely sensory. We had here a beginning thalamic syndrome which was practically never found in hysterical states. This patient further had a sinus disturbance on the right side which rhinologists had diagnosed as specific or tuberculous.

Dr. Frederick Tilney of Brooklyn read a paper on "New Clinical Instruments for Measuring Muscle Strength and Tendon Reflex Threshold," with lantern-slide illustrations, and showed two new clinical instruments of precision for measuring muscle strength and tendon reflex threshold.

Dr. Louis Casamajor said the devising of these instruments of precision by Dr. Tilney was a step in the direction of placing neurology on a more exact scientific plane. The dynamometer he had found of extreme value in testing the strength of certain groups of muscles, particularly in cases of anterior poliomyelitis and in watching the return of the muscle function in traumatic brachial palsy. With this instrument we could detect the earliest return of function and could base our prognosis upon exact scientific measurement rather than upon pure guesswork. He had also found it of value in drop foot and other conditions and it had made a strong impression upon him as being something more accurate than anything of the kind we have had before.

Dr. Timme said the stereopticon pictures of the tendon reflexes shown by Dr. Tilney were an excellent illustration of the accuracy of this instrument, as compared with the older methods. Instead of adding to the history of our case that the "reflex was still deficient" or an equally unsatisfactory expression, we had here an instrument which gave us in exact terms the amount of improvement or otherwise that had taken place.

WILSON'S LENTICULAR DEGENERATION

By Charles E. Nammack, M.D.

A boy, 20 years old, was admitted to Bellevue Hospital on June 20, 1914. He had never had any regular occupation. His father died at the age of 42 of glandular tuberculosis; his mother at about the same age of pulmonary tuberculosis. His parents were not consanguineously related. The mother had seven children, the patient being the last child. Of the seven children, four were living and in good health. One brother died of pulmonary tuberculosis. The patient stated that so far as he knew, no one in either branch of the family had a condition similar to his. The family history was negative as to gout, rheumatism, syphilis, alcoholism or nervous affections. One of his brothers was married and had one child, in good health. There was also a married sister with two children in fair health.

This patient attended school until he was unable to stay in the class. He smoked cigarettes and occasionally drank a glass of beer. He had the usual diseases of childhood and at the age of five years he lost his speech and the power of walking, being obliged to creep around, but he was able to go to school at the age of ten, and left six years later, in 4-a grade. He states that he was always near the head of his class and liked to study. He had a bad temper and would get into quarrels with the other boys. He left school at the age of 16 because his left arm began to shake so that he could not hold objects in it. After he left school he remained at home, occasionally going out with draymen, etc., but he had never had any regular work.

The first symptom of the disease occurred when he was six years old, when it was noticed that his left hand trembled considerably and that his right leg slowly turned in, as the patient expressed it. The foot finally got into such a condition that he walked on the side of the foot and had to catch hold of bed-posts, etc., to support himself. He was taken to the family physician, who said that he had St. Vitus' dance. There was no history of epileptic fits or periods of unconsciousness. Gradually, the

tremor of the hand became more aggravated, the condition of the foot became worse, and his speech became so nasal in quality that it was difficult to understand him. When he started to do anything he had to wait a few minutes and then quickly arose; as soon as he got up he would



FIG. 1. Case of Lenticular Degeneration.

start swaying around and would have to take hold of some support to pull himself along. If he was in the open, without support, he would throw himself forward and drag or slide his feet. After he started walking he got along very well unless he stopped; states he could walk across the Brooklyn bridge providing he did not stop, but kept right on. The muscles of the arm and trunk were remarkably well developed.

The patient's facial expression was fixed; the mouth was large and drawn, and when walking or doing any form of exercise he protruded his tongue. He understood everything that was said to him and was usually in a jolly mood, although he had a bad temper. His intelligence was about on a par with that of a boy of twelve or thirteen. He was able to



FIG. 2. Case of Lenticular Degeneration.

dress and feed himself; only it took him longer. His condition had apparently remained unchanged for several months; at least, it did not appear to differ physically or intellectually since the date of his admission on June 20, 1914.

The patient was well developed and nourished. His movements were

jerky; that is, his head and arms would jerk while his spine became rigid with a functional anterior curvature, with talipes equinovarus of the left side. As he bent forward, the right foot was thrown out in an inverted position, and the left foot was drawn by sliding, in the meantime the head, arms and pelvis going through jerky motions. The speech was long and drawling, making it difficult to understand him. Swallowing was normal. The tremor of the hand was coarse. The grip was good; the knee and ankle jerks were exaggerated; sensations appeared to be normal. The Wassermann test was negative. There was atrophy of the muscles of the right leg, hip and calf, with compensatory hypertrophy of the other muscles of the body.

Dr. Nammack said that when this boy was about ten years old he was treated at the Cornell Dispensary, without diagnosis. About two years later he was under treatment at the Vanderbilt Clinic; diagnosis withheld. When he was admitted to Bellevue Hospital in June, 1914, the case was regarded as a puzzle until Dr. Foster Kennedy called attention to Wilson's article on lenticular degeneration, when it was placed in that category. So far as he was aware, Dr. Nammack said, this was the first case of Wilson's lenticular degeneration to be shown in this city. One case had been reported in Philadelphia.

Dr. Foster Kennedy said that while he was inclined to regard this case as an example of Wilson's lenticular degeneration, he recognized the fact that there was considerable scope for argument in the diagnosis. In several important respects, this case did not absolutely conform with the clinical description given by Wilson in his paper. One was the comparatively slow progress of the disease in this case—its long duration. Then again, there was the lack of emaciation and pyrexia and the absence of any evidence of liver disease. Of course, it was fair to presume that the lenticular nucleus could be diseased without visceral involvement and give rise to a syndrome somewhat dissimilar from that described by Wilson, but as the case stood now, it did not conform in the above particulars with the original description of Wilson's disease, which was regarded by him as a clinical entity, dependent on toxic causes, and the main feature of which was involvement of the liver associated with degeneration of the lenticular nucleus. It was unfortunate that the name adopted by Wilson, *i. e.*, progressive lenticular degeneration, did not suggest the presence of concomitant liver disease. Whether or not the liver was cirrhotic in the case shown by Dr. Nammack could not be definitely stated at this stage. In only one of Wilson's cases was there jaundice.

Dr. J. Ramsay Hunt said that he had become very chary in making the diagnosis of Wilson's disease, since an experience with a case which had been under observation for a number of years at the Montefiore Home in which the clinical picture was in many respects typical of this condition. The progressive rigidity and the terminal symptoms corresponded in almost every particular with Wilson's description of lenticular degeneration. At the autopsy, however, the liver was found to be perfectly normal and careful serial sections of the lenticular nucleus showed no macroscopic lesions. Some histological studies were now being made in this case which were not yet complete.

The clinical picture of Wilson's disease, therefore, may be very closely stimulated by a clinical type which is probably a juvenile paralysis agitans.

In the case presented by Dr. Nammack, the symptoms which are now of 14 years' duration, seemed to have been consecutive to an acute

central affection of early childhood. It is not unlikely that the entire clinical picture is simply the result of a polioencephalitic process in early life, and not a progressive degenerative condition.

Dr. Smith Ely Jelliffe said that the bilateral character of lenticular degeneration as originally described by Dr. Wilson assumed importance. This patient did not show a bilateral tremor. The patient had athetoid movements of the left upper extremity, and palsy and spasm of the right leg, and the speaker said he was inclined to agree with Dr. Hunt that we had here the results of a polioencephalitis occurring in youth, with athetosis on one side and spasm on the opposite side. In the cases described by Wilson the tremor was invariably bilateral. In some of the patients which were presented at meetings of this Society by Dr. Jelliffe as probable cases of multiple sclerosis with paralysis agitans like syndromes, the possibility of Wilson's degeneration was considered and the relationships to this disease taken up. He thought that the diagnosis of lenticular degeneration could not be successfully maintained in this case on account of the absence of any hepatic involvement, the long duration of the disease, the non-bilaterality of the symptoms, and the doubtful clinical findings.

Dr. Nammack, in closing, said the possibility of an old cerebral hemorrhage, with post-hemiplegic athetosis, was considered in connection with this case, but subsequently rejected. The patient's symptoms had not been noticeably progressive since his admission to the hospital four months ago.

As regarded the long duration of the disease in this case, and its bearing upon the possible diagnosis of Wilson's disease, Dr. Nammack said there was considerable difference of opinion among those who had reported cases of lenticular degeneration since Wilson's original description. Wilson divided his cases into two groups, *i. e.*, acute and chronic, the duration of the latter averaging four years. Sawyer's case lasted seventeen years, but when Wilson saw this case he threw it out. None of the cases reported in this country entirely agreed with Wilson's description. While this case was somewhat analogous to the juvenile type of paralysis agitans, there was enough difference to separate them clinically.

THE TREATMENT OF SYPHILIS OF THE NERVOUS SYSTEM WITH SALVARSAN IN THE FRANKFURT AND HAMBURG CLINICS

By F. J. Conzelmann, M.D.

Dr. Conzelmann described in detail the methods employed in the treatment of syphilis of the nervous system with salvarsan in the two representative clinics in Germany—the Dreyfus clinic at Frankfurt and the Nonne's clinic at Hamburg.

At the Dreyfus clinic the first idea of salvarsan was conceived, and here the great master Ehrlich visited the clinic, rendered individual supervision and offered suggestions. One was impressed with the well-planned and orderly arrangement of the clinic, and the feature that stood out most prominently was that Dreyfus did not treat the patients as merely cases, but as individuals. He first selected the type of the disease; secondly, he gave the intensive treatment of salvarsan and mercury;

thirdly, he adapted the dose of salvarsan or mercury to the individual and was guided by the constitutional reaction; and, fourthly, the treatment was continuous, with definite periods of intermission.

Ehrlich and Dreyfus preferred old salvarsan to neo-salvarsan, and used the latter only on certain indications, especially when a mild, non-irritating action was desired; nevertheless, the initial treatment was frequently begun with neo-salvarsan. The concentrated solution of salvarsan was always used, and was prepared as follows: The dose of old or neo-salvarsan was dissolved in 35 c.c. of double distilled water; then the sterile sodium hydroxide was added and the solution injected with the syringe directly into the vein.

A searching physical examination of the patient preceded every course of treatment with salvarsan; in fact, it determined the final treatment. Patients suffering from syphilitic affections of the nervous system soon after the primary infection were as a rule strong, robust individuals, and could tolerate a much larger dose than those in whom the infection had been present for many years. If the patient suffered from gastric trouble, a thorough stomach analysis was made and the condition treated before giving salvarsan. In patients over 60 years of age, salvarsan was given with a great deal of care. In cases with tuberculosis, chronic heart disease or nephritis, the pros and cons were carefully considered.

A course of treatment lasted six or eight weeks, during which time the patient received from three to six grams of old salvarsan, or from four and a half to nine grams of neo-salvarsan, with from six to twelve mercurial injections of 0.02 to 0.05 of a 40 per cent. calomel solution, or the same dose of some especially prepared oily solution of mercury. Patients, after receiving a dose of salvarsan intravenously, were kept in bed for twenty-four hours; if, at the end of that time, there was no feeling of illness nor rise of temperature, they were permitted to get up. Two examinations of the urine weekly were considered imperative.

After the physical examination of the patient, which included lumbar puncture and a Wassermann test of the blood and spinal fluid, had been completed, the diagnosis of the stage of the disease was agreed upon, and a carefully planned, systematic course of treatment was begun. The approximate outline of such a course of treatment in early brain syphilis was as follows: A course of mercurial treatment consisting of five injections of 0.02 to 0.05 c.c. of a 40 per cent. calomel solution every third day. If, at the end of this time, the patient showed no febrile reaction to the injections of mercury, the injections of salvarsan were begun. The mercury injections were continued every third day, and a concentrated solution of 0.15 grams of neo-salvarsan was injected directly into the vein with a syringe. If this dose produced no reaction, the patient received a second dose of 0.3 gram on the following day; no untoward symptoms developing, he received 0.45 the third day and 0.6 on the fifth day. This was continued every other day until the patient had received from one and a half to two grams of neo-salvarsan. Dreyfus began the treatment with old salvarsan, generally starting with a small dose of 0.1 and never going beyond 0.4. The dose was repeated every second day. If at any time during the treatment the patient had a febrile reaction after the injection, the treatment was discontinued and not resumed until the patient was free from fever for at least two days.

A favorable prognosis was given in early brain syphilis—in fact, a positive cure was promised—to all who returned for reexamination and

took the treatment if the physician advised it and considered it imperative. In the majority of cases of late syphilis of the cerebrospinal system the prognosis in the Dreyfus clinic was generally favorable for improvement, but not for cure. In tabes the patients were treated with salvarsan alone for three weeks, giving one injection of salvarsan of 0.1 to 0.2 gram every other day. At the end of that time the treatment was combined with mercury. As a rule, Dreyfus preferred mercurial inunctions in cases of tabes, but often used the mercurial salicylate. With this form of treatment the patients improved rapidly, gastric and lancinating pains disappeared and the ataxia and visceral symptoms improved. Tabetic cases were always emphatically advised to take a course of treatment every three months until four courses were taken.

In the intraspinal administration of salvarsan, Dreyfus used Genenrich's modification of the Swift-Ellis method. He dissolved 0.15 gram of neo-salvarsan in 300 c.c. of sterile salt solution, and of this solution 5 c.c. were injected intraspinaly after the withdrawal of an equal quantity of spinal fluid. His results in initial lues cerebri were very encouraging, while in tabes the results of the intraspinal method of treatment were symptomatic but nevertheless very striking. Dreyfus had no cases of general paresis in his ward.

Since February, 1914, Dreyfus had used two new preparations of salvarsan from Ehrlich's laboratory: the one was copper salvarsan; the other sodium salvarsan. Both could readily be dissolved in water and did not entail the manipulation required in the preparation of old salvarsan. The solutions were given in concentrated form directly into the vein with the syringe. With this mode of administration three or four cases could easily be treated in the course of half an hour.

In Nonne's clinic at Hamburg, every patient suffering from syphilitic or meta-syphilitic disease of the nervous system received a course of mercurial inunctions, irrespective of the stage of the disease. For purpose of treatment, Nonne had his tabes cases divided into groups: one group received mercury inunctions; another group mercury inunctions and intravenous injections of salvarsan, a third group mercury inunctions plus intraspinal injections of salvarsan and a fourth group received no treatment whatsoever. In cases of general paralysis he used the mercurial inunctions and intraspinal injections of salvarsan; in cases of cerebral lues he preferred to give mercurial inunctions, with intravenous injections of salvarsan. He employed von Schubert's modification of the Swift-Ellis method. In his tabes cases Nonne got improvement, symptomatically, by the intraspinal method, but in some cases the bladder symptoms were very much worse after the treatment. In two cases the gastric crises returned after a month. He also claimed symptomatic improvement in his cases of general paralysis.

In concluding his paper, Dr. Conzelmann said there was no doubt that no effort was spared in the laboratories and clinics in Germany to develop the science of saving human life. He was still in Nonne's clinic when war was declared, and before he left it appeared as if they had not neglected to develop the science of destroying human life, but on this he would not venture an opinion. He was much in the condition of a depressed maniac who could form no judgment, come to no conclusion and reach no decision.

Dr. J. F. Terriberry said he had under his observation at the present time a man about 30 with tabes who was so much disturbed by the old-

time anti-syphilitic treatment that all treatment was discontinued. Under this let-alone method he had remained comparatively free from symptoms during the past year, and excepting for a slight difficulty in locomotion he seemed to be in perfect health. We must not underestimate nature's restorative powers under good hygienic and dietetic conditions.

Dr. Conzelmann, in reply to a question, said that so many of Nonne's cases of tabes got along well without any treatment aside from rest in bed that Nonne was rather skeptical in regard to the value of the intraspinal injections in cases of tabes.

Translations

VAGOTONIA

A CLINICAL STUDY

BY PRIVATDOZENT DR. HANS EPPINGER AND DR. LEO HESS

OF VIENNA

TRANSLATED BY WALTER MAX KRAUS, A.B., M.D., AND SMITH
ELY JELLIFFE, M.D. PH.D.

(Continued from vol. 42, page 50)

Let us take spastic constipation as an example. This may be associated with symptoms, which are limited to the pelvic nerve alone. One often hears that these costive patients have erections, spermatorrhea, or pain in the sphincters, associated with the attempt to defecate. Could we not suppose that the entire pelvic nerve is hyperirritable under these circumstances, and that it is stimulated by the act of defecation, so that, by an overflow of energy, the branches other than those supplying the rectum, are involved. Furthermore, we have seen a group of cases in which epiphora, salivation and conjunctival asthma, as Rosenbach has described it, were associated, while in organs supplied by other branches of the autonomic no signs of stimulation could be found. In such cases we suppose that certain poisons, circulating in the body have the same partial action as has been found to be the case with some drugs.

Let us sum up in a few sentences what has been said in the last two chapters: (1) Vagotonia is a functional increase of tone in the autonomic system. (2) This increase may affect nearly all or but a few of the branches. (3) The origin lies in a latent increase of function and this permits stimuli to act more readily than on a nervous system in which no such increase exists. (4) Vagotonics, therefore, are more responsive than individuals having a normal vegetative system.

8. COMBINATION OF VAGOTONIA WITH OTHER DISEASES

In the foregoing chapters we have attempted to show that many people have a constitutional condition which makes a visceral nerve—by this is to be understood the entire autonomic or extended vagus, more irritable than its antagonist, the sympathetic. When we consider that these two nerve plexuses control all visceral organs, we cannot fail to realize that a disturbance of one of them will produce anomalous conditions in many organs. On the one hand this constitutional condition may be regarded as stationary and as of long duration,—on the other hand, there are periods in which the condition will be exaggerated and more susceptible to irritation. This fact may well be correlated with the occurrence of various diseases at certain periods of life. We recognize various periods, which dispose to certain diseases, as old age, youth, climacterium, puberty, and menstruation, and we know that during these periods, certain groups of symptoms take the foreground. It seems worthy of question whether or not the constitutional state, which we have described does not also influence certain diseases in a similar way. We have been able to show that spastic states in a limited part of the autonomic system occur in individuals having precisely this constitutional condition. Our attempt to draw other pathological conditions into correlation with the constitutional state we have described must not overshadow the fact that we do not intend to divide all individuals into two classes: the vagotonic and the sympathicotonic, but shall base whatever conclusions we may reach, on cases in which there have been well-defined functional disturbances in two or three parts of the autonomic nervous system. Furthermore, the cases must have shown these disturbances not only before, but also after the disease. We hope to be able to show in what follows that those characteristics of a particular course of a disease, which heretofore have been ascribed to individual idiosyncrasy, are in reality due to a lack or excess of a definite nervous disposition of the organism

Pain is the simplest example of the rôle which nervous components of a syndrome play in its symptomatology. In the same syndrome various individuals may have the intensity of a certain pain vary to a great degree. Certain individuals are not susceptible to the action of cocaine, and the admission that it has not

acted in the right place must be made. Analogously we must admit that poisons produced by certain diseases do not always act on the same parts of the body in each case. For this reason, certain very definite, well known symptoms often are in the background, while others are so conspicuous that the classical picture of the disease is entirely effaced.

For example, should a vagotonic get a gastric ulcer of organic origin, one can readily see that the stimuli from the lesion will pass along the more readily irritable vagus. Thus we shall find in vagotonics with gastric ulcer, a hyperacidity and both increased tone and peristalsis of the stomach, combined with the symptoms they produce. Furthermore cases of gastric ulcer will show signs of autonomic irritation in other parts than those directly affected. Thus we find bradycardia²⁶ as well as the cardinal symptoms of ulcer, hyperacidity, and great pain combined with retching and a tendency to spastic constipation. The symptoms may vary according to the locality of the ulcer. In some cases of ulcer one finds signs of hour-glass stomach. At operation or at autopsy this diagnosis is not confirmed.

When it is considered that X-ray signs of hour-glass stomach are found in simple vagotonics, there is no cause for wonder that when an ulcer exists, this can also occur, and that these signs of hour-glass stomach are due to an increased tone of the gastric musculature. This in turn is made clearer upon the basis of an increased tone in the autonomic system. On the other hand, it is known that there are cases of gastric ulcer in which no signs of gastric hyperacidity occur, and which are unaccompanied by pain, the only clue to their existence being the presence of occult blood in the stools. Some are found only at autopsy. It seems very significant that signs of general vagotonia are never found in such cases. Cases of gastric ulcer have been seen in which the hydrochloric acid value was not at all increased. These constituted the most difficult group to diagnose from carcinoma, an observation of the greatest interest, since these are cases which tend to develop true neoplasms—callous ulcers. When these facts are considered the question arises whether the various clinical courses of such diseases as Cholangitis, Cholelithiasis and Nephrolithiasis are not in some way related to constitutional variations.

²⁶ Neusser, *Ausgewählte Kapitel der klinischen Symptomatologie und Diagnostic*, 1 H. 1904.

Certain cases of gall-stone disease particularly show symptoms which are due to autonomic stimulation. Since this exists not only before but also after the attack, the assumption of a latent constitutional disorder, which alters the course of disease, is well founded. Cholecystitis is often associated with hyperacidity. The vomiting and diarrhea may be referred to the attack, but all of these also presupposes a certain predisposing irritability of the organs involved. We believe that the spastic state, which begins in the gall-bladder and spreads thence to neighboring organs, causing vomiting or diarrhea, may also pass over into the gall passages. Only in this way is it possible to account for the transitory jaundice which occurs in cases of acute cholecystitis, in which there is no reason to believe that a stone is present to cause the jaundice mechanically. That the stone should be forced by the colic into the ductus choledochus and should then fall back after the colic is over, seems very improbable. Finally we wish to call attention to the inconstancy of the relation between bradycardia and jaundice in these cases and to the additional fact that the bradycardia may not exist in old people. In the young as well great variations may be observed, so that here, too, we must assume some predisposition of the cardiac vagus. If on the one hand, gastric ulcer and some cases of gall stone colic are related to autonomic irritation, it would seem on the other hand that the diminished gastric acidity and mobility, and the dry skin and mucous membranes of carcinoma signified a diminution in the activity of the autonomic. In this connection it seems significant that we found that adrenalin glycosuria was almost never absent in the more severe types of general carcinomatosis, while pilocarpin never gave strong reactions. Thus the question is raised whether perhaps the nervous constitution does not play a rôle in the occurrence of tumors. The question also arises,—does the constitutional state of the individual cause a definite course in such autonomic diseases of the nervous system as *tabes dorsalis*.²⁷

It is well known that there is a group of cases of this disease which begin with visceral disturbances, crises, as the most prominent symptoms, nor is it infrequent to find cases in which these crises are entirely lacking. It is not difficult to show that the crises themselves are signs of autonomic irritation. The narrowing of the pupil at the onset of the attack, the subsequent gastric

²⁷ Eppinger and Hess, *Wiener klin. Rundschau*, 1909, No. 47.

hypersecretion,—sometimes associated with hyperacidity, the increased peristalsis and sweating, which may occur alone or at the height of some other crises, the epiphora, laryngeal and rectal crises, all show that at the height of the attack the autonomic is in a state of irritation. The fact that the spastic states are replaced in the later stages of the disease by their opposites (paralysis of the recurrent laryngeal, marked gastric and intestinal atony, anacidity, and even incontinence of feces and urine impotence, etc.) leads us to believe that what were originally irritative states of the autonomic have been replaced by paralysis of those centers which originally caused the irritation. A proof of this for us is in the discovery that in many severe cases of tabes, degenerations were found in the trunk and nucleus of the vagus. It may be permitted us on these grounds to assume that the toxic agent of tabes dorsalis, in addition to attacking the posterior spinal cord columns, has also a specific affinity for the autonomic system, since it first irritates and then paralyzes it. It is difficult to say whether or not the crises, regarded as spastic states, are merely states of irritation superimposed upon vagotonia. It is, however, striking that cases without crises, do not show signs of general vagotonia. A further argument in favor of our hypothesis is that those signs which are so often found associated with vagotonia, as for example, status thymicus and lymphatism, were never found in cases of tabes without crises, since according to Edinger's theory of the consumption of neurotropic poisons, they first affect those nerves which are functionally most active, we might, by analogy, consider the possibility that the already over-irritable autonomic system would be most prone to take up the toxic agents of tabes dorsalis.

The following case may be taken as an example of the predisposing affect, which pre-existing vagotonia has upon the occurrence of crises in tabes:

A. F., a merchant, has complained since his youth of nervous stomach trouble. He took soda for its relief. Fifteen years ago a gastric analysis was done and showed hyperacidity. He has been costive for many years, going to stool but every three to four days. The stool is very small in size, and often mixed with mucus. He has a tendency to salivate and sweat at the same time. Ten years ago the patient was infected with syphilis. This was followed by a course of mercurial inunctions. The first signs of gastric crises occurred over three years ago. Three years ago the symptoms increased so that he had continuous vomiting and marked gastric pains for two to three days of

each week. Even when he had not taken food, the patient vomited large amounts of an acid, bile-stained fluid. As the increase in frequency of these attacks continued, the patient came to the clinic for advice. Objectively here were absence of the light-reflex, permanent absence of the knee-jerks, and a moderate anesthesia in the region of the right nipple. The "Head zone," corresponding to the stomach, showed marked hyperalgesia. During the crises, the blood pressure fell from the normal 105 mm. Hg. to 70 and even 60.

Basedow's disease is another example of the significance of vagotonia in understanding pathological conditions. It will be very difficult to account for all the symptoms of this disease on the basis of one cause, if one takes the point of view that they are referable to nerve irritation. Symptoms which are referable to sympathetic stimulation are found associated with others undoubtedly referable to autonomic stimulation. The cardinal symptoms themselves, exophthalmos and tachycardia on one hand, sweating and diarrhea on the other, indicate stimulation of both parts of the vegetative nervous system. In another article we have attempted to show that one explanation will not account for all the accessory symptoms, but that in the individual, both autonomic and sympathetic stimulation are combined, even though this may be but a transitory matter. On the other hand, we have found typical cases of Basedow's disease, in which all the symptoms were due to stimulation of one of the two nervous systems. The diagnosis in these cases was made certain by the increase in the level of metabolism shown by a marked degree of emaciation in spite of a sufficient diet. On the basis of these cases, we divided Basedow's disease into a vagotonic and a sympathicotonic type. Characteristic of the first type were: (1) relatively slight increase of the pulse rate, (2) subjective cardiac symptoms, (3) marked v. Gräfe's sign, (4) wide lid-slits, (5) absence of Moebius's sign, (6) very moderate grade of exophthalmos, (7) marked epiphora, (8) excessive sweating, (9) profuse diarrhea, (10) symptoms of gastric hyperacidity, (11) eosinophilia, (12) disturbances in the rhythm and mechanism of respiration, (13) absence of alimentary glycosuria.

In the sympathicotonic cases there were (1) marked exophthalmos, (2) No. v. Gräfe's sign, (3) positive Löwi's phenomenon, (4) marked Moebius's sign, (5) frequently dry eyes, (6) a high degree of tachycardia with no subjective symptoms, (7) no sweating, (8) no diarrhea, (9) marked falling out of the hair, (10) tendency to rises in temperature, (11) no eosinophilia, (12) no

respiratory disturbances, (13) alimentary glycosuria. We were able to establish the antagonism between adrenalin and pilocarpin in these cases. The number of cases in which one or the other system was excessively irritated was, however, very small. Those which were of the sympathetic type showed but little reaction to pilocarpin, while those which were of the vagotonic type showed no glycosuria after adrenalin. These types, which are not always constant, one passing into the other in various phases of the disease, led us to believe that at the root of the matter lay a difference in the make-up or constitution of the individual. By this we mean simply that those who are vagotonic before acquiring the disease, will be mainly of the vagotonic type during it, while those who were previously of the sympathicotonic type will have symptoms corresponding to those produced by sympathetic irritation. Furthermore, should the individual have been neither vagotonic nor sympathicotonic before the onset of the disease, the thyroid secretion, since it affects both symptoms, will cause symptoms of irritation of both.

It is worthy of mention that the one case, which was of the pure sympathetic type showed practically no thymus nor any lymphatic tissue. To this we would add that in another case, which had sympathetic symptoms for a long time, but developed in addition some vagotonic symptoms just prior to death, showed a large thymus and well-developed lymphatic system at autopsy. In spite of this, we believe that those who are vagotonics before acquiring a Basedow, will show during the disease the symptoms produced by the action of those components of the thyroid secretion, which acts upon the autonomic nervous system. By this we do not wish to imply a condition of dysthyroidism, but that these individuals do not react to those constituents of the thyroid secretions, which stimulate the sympathetic, just as certain individuals do not react to adrenalin. The fact that both at the height of a severe case of Basedow and during the course of a very mild one there may exist severe symptoms of irritation of both systems may be interpreted as the weak point in our idea of their genesis, since we have advanced the viewpoint that if the vagus is strongly stimulated, it is scarcely possible that there should exist a simultaneous high degree of stimulation of the sympathetic.

Observations upon those afflicted with mental disease have shown that when it is most severe there is a disturbance of the

equilibrium of the vegetative nervous system. Thus we can draw an analogy between Basedow's disease and central nervous system disease and say that where either exists the equilibrium will be disturbed. We observed that in many cases of catatonic or maniacal excitement there occur in quick succession strong glycosuric reactions to adrenalin, followed by marked responses to pilocarpin. We never saw so high a degree of glycosuria as in those having mental disease (early stage of dementia præcox), nor such marked responses to pilocarpin. After the usual dose of pilocarpin (gr. 1/6), some cases had such severe salivation and epiphora, besides a discharge from the nose, that there was almost a continual stream from both nose and mouth. These were the cases which, after the administration of pilocarpin, usually reacted with spermatorrhea, vomiting, severe sweating and urinary urgency.

We often found a reaction of both parts of the vegetative nervous system in diseases of the spinal cord, as multiple sclerosis or transverse myelitis.

The fact that during the psychic disturbances of mental disease over-irritability of both parts of the vegetative nervous system could be demonstrated, permits us to give the same fact more consideration in Basedow's disease.

In looking over our case histories, we found that the group of cases, which showed alternately signs of irritation of the two systems were not complicated by mental disturbances, while those which showed these signs simultaneously often were complicated by psychical disturbances of one sort or another.

In investigating psychoses we found individuals who did not react to the usual dose of sympathicotropic or vagotropic substances. Since these cases were mainly of the depressed type, we feel that our observations are of some significance in the consideration of the pathogenesis of certain psychoses.

The absence of central control may play a part in Basedow cases as well as it does in mental disease with excitement. This point of central control brings us naturally to the idea that under physiological conditions, the central nervous system exerts a definite influence over the antagonistic parts of the vegetative nervous system.

(To be continued)

Periscope

Allgemeine Zeitschrift für Psychiatrie

(Band LXX, Hefts 3 and 4)

1. Privy Councillor Dr. Heinrich Schüle. A Complimentary Notice about Dr. Schüle who has just celebrated the 50th anniversary with the Iltenau Asylum.
2. The Pathological Histology of Korsakow's Psychosis. ERNST THOMAS.
3. Psychoses in Twins. DR. SCHULTES.
4. Contribution to the Diagnostic Application of the Wassermann Reaction in Psychiatry. R. BUNDSCHUH.
5. Psychically Abnormal Tramps. HANS ROEMER.
6. A Contribution to the Psychopathology of Family Murder by the Insane. WALTHER HASSMANN.
7. The History of the Foundling Asylum, Madhouse and Hospital, also of the House of Correction at Pforzheim. W. STEMMER.
(Unsuitable for Abstract.)
8. A Contribution to the Understanding of the Animal Mind. LUDWIG WILSER.
9. The Development of the Building of Insane Asylums. MAX FISCHER.
10. The Question as to the Basedow Psychoses. ERNST COLLA.
11. The Forensic Importance of Chorea. DR. HORSTMANN.
12. Lunacy Laws in Servia. W. M. SUBOTIC.
13. The Deaf Mute as a Witness in Court. AUGUST HEGAR.
14. Self Mutilation in General Paresis. H. BRASSERT.
15. Epilepsy and Sedobrol. DR. BÖSS.
16. The Rulers of the Julian-Claudian Imperial House in Historical, Genealogical and Psychiatric Light. ERNST MÜLLER.
17. Admissions to the Curative and Custodial Asylums as well as to the Psychiatric Clinics of the Grand Duchy of Baden, from 1826 to 1910. HERMANN HERR.

1. Of local interest only.

2. *Korsakow's Psychosis*.—In volume 67 of his journal the author reported his findings in two cases of Korsakow's psychosis and here adds those in another examined by more specific methods not used in his former cases. The case was that of a man 49 years old whose father was alcoholic and one paternal aunt insane, whose urine had contained some sugar and who, having shown the typical Korsakow's symptom-complex, died of myocardial degeneration. The nerve cells, examined by Nissl's method, showed various degenerative changes. There was degeneration of fibers especially of the tangential layer, the fibrils by Bielschowsky's method showed degeneration and thinning of the fibril network in some places, while the neuroglia by Weigert's method, showed quite general increase in the diseased areas. The sections were taken from the first and second frontal, the anterior central convolutions and the region of the calcarine fissure. The degenerative changes were pretty generally distributed though least marked about the calcarine fissure. The fine vessels of the cortex showed no

changes though the larger ones had their walls somewhat thickened. The author recapitulates as follows: (1) This last investigation confirms in the main the former findings both of the writer and of Vollrath and Meyer. (2) He agrees with Meyer in considering degenerative processes as the characteristic disease process. For this speaks the circumstance that in cases running an acute course the degeneration is much more rapid than in chronic cases. (3) The most marked alterations were found in the anterior central convolution, less change in the first frontal while the occipital region was least affected. (4) In the affected regions the third to the sixth layers were more affected than the first and second layers. The tangential fibers were less affected and there was less proliferation of neuroglia than is usually the case in paresis. (5) In contradistinction to this last disease there is less shrinkage of the cortex in general.

In general it may be said that corresponding to the anatomical findings there is scarcely such profound mental destruction as in general paresis. The investigations of Gregor have shown that in spite of great disturbances of comprehension and memory in Korsakow's patients, they still retain a certain ability to learn, differing in this from paretics. Since in his cases the two outer cortical layers were found least affected he raises the question as to whether the better mentality as compared to paretics is not connected with the preservation of these layers. According to Nissl's investigations the deeper layers are concerned in localization, the more superficial in other functions, presumably in association. This article is illustrated by some good reproductions of photomicrographs.

3. *Psychoses in Twins*.—Mental disease in twins is of particular interest, since here we have two individuals who have been presumably exposed to exactly the same prenatal influences. To the limited number of cases already reported the author is able to add five additional sets of cases from the Illeau Asylum. As to the clinical form of the psychoses, in observations I and III it was manic depressive insanity. In I both twins were afflicted with typical circular insanity in which manic, melancholic and nearly normal phases alternated. In observation III there was also manic-depressive, but in one case it was complicated by alcoholism. In observation V, in spite of some traits in common, depending probably upon a common constitutional basis there was in one twin manic depressive, in the other hysterical insanity. In observations II and IV dementia præcox was present. In psychoses in twins the factors of heredity and induction come chiefly under consideration. The factor of induction can never be completely excluded. It is favored by growing up together in close intimacy and the action on both individuals of the same external influences about the same time of life. In the first observation the earliest period of excitement coincided with menstruation, which of course does not always coincide exactly in time in two persons. Later in the two sisters the opposite phases of the disease occurred nearly always about the same time, so that induction can be nearly excluded. In observation II psychic infection may well be considered as an exciting cause. In observation III the outbreak of the disease was widely separated in point of time in the two twins. It can be traced in each case to a somatic exciting cause, in one brother to nephritis, in the other to trauma. The pair were separated, each being married and living with his family. In observation IV the close interdependent intimate relation in thought and pursuit makes the influence of the inductive factor seem quite probable. In observation V the twins had lived apart

from earliest childhood and up to the outbreak of the psychoses had had no close relations. Later, however, worry about the sick sister may have acted as an exciting cause for the depression of the other sister. In all the cases there was more or less hereditary predisposition to psychoses, in fact a converging heredity. As to the other children of the family, in observation I, only the twin sisters were insane while all the brothers remained healthy. The same thing was the case in observation IV. In observation II the twins were the only children. In observation III besides the twin brothers one other brother was insane, while the sister remained healthy. In observation V besides the twin sisters one other sister was insane while all the brothers in spite of some peculiarities of character remained sane and led useful lives. In four of the sets of twins the resemblance between the two was striking, both in the mental and in the physical makeup. In V alone the similarity was less marked. In the like twins there was a like clinical form of disease, in the dissimilar ones a different psychosis in each. The like twins can be readily supposed to have originated from a single ovum with two nuclei, the unlike twins from separate ova fecundated at the same time.

4. *Diagnostic Application of the Wassermann Reaction in Psychiatry.*

—As method this author finds the original recommendation of Wassermann most satisfactory, no other antigen working so well as extract of the liver from a syphilitic fetus, the watery extract being in general preferable to the alcoholic. In 71 cases of general paresis in which the blood serum and the spinal fluid were examined, 0.2 c.c. of the latter being used, the result was uniformly positive and in general equally strong in each. The material included paretics at all stages. One juvenile case developed upon the basis of congenital syphilis reacted with equal positiveness. In one case which had lasted four and a half years the reaction in the fluid was somewhat weaker than that in the blood, while in another of 16 years duration which had remained for years in a stationary condition of dementia the reaction was equally positive in both blood and fluid. In four cases in which there was marked remission the reaction was inhibited from the start. The author agrees with Plaut that the Wassermann reaction is always positive in the earliest stages of paresis and is of the greatest value in its differential diagnosis. In general he did not find that the Wassermann reaction was to any noteworthy extent influenced by the treatment of paretics with nucleinate of sodium, mercury, and salvarsan. Still he would recommend caution in giving a positive opinion in cases which have had salvarsan treatment, since while he has not found the reaction made negative he has observed that it was decidedly weakened. Nonne's globulin reaction was positive in all of 54 paretics upon whom it was tried, but he could not find that its intensity was parallel with that of the Wassermann reaction. Although some doubt has been thrown upon the matter, our author is of the opinion that by the use of the "Auswertungs" method (*i. e.*, the application of the Wassermann reaction to fractional portions of the spinal fluid, in this instance from 0.2 to 1.0 c.c.) we are in a position to differentiate cerebro-spinal syphilis, on the one hand, from non-syphilitic psychoses in syphilitics, and on the other from general paresis, since he has found that in the last disease there is without exception a positive reaction to the usual Wassermann procedure even when 0.2 c.c. of spinal fluid are used, the cases of cerebral syphilis failing to react with 0.2 c.c. while they give a positive reaction when the fluid is raised in amount to 1 c.c. and syphilitics who have

no syphilitic involvement of the central nervous system while showing no reaction from the spinal fluid give a positive one from the blood. As illustration the author gives the histories of 9 cases of cerebral syphilis in which a positive result by the "Auswertungs" method enabled him to make diagnoses from general paresis on the one hand and from non-syphilitic psychoses on the other. Since the Wassermann reaction has become well known among the laity, he has been frequently requested by the relatives of paretics to apply this test to the wives and children of such subjects, and thinks that this has become a social duty of the psychiatric-serologist. Of all wives examined, 6 gave a negative, 5 a positive reaction. He also examined 10 children, some of whom showed some weakness, anemia and slight intellectual and affective disturbances. In all of these, the mothers having given negative reactions, the result of the Wassermann test applied to the blood was negative.

5. *Psychically Abnormal "Tramps."*—A statistical study of the material admitted to the psychiatric clinics in Heidelberg and Freiburg and into the public asylums, Ilenau, Pforzheim, Emmendingen, and Wiesloch, which the author thinks will give a fair representation of the insane among the tramps in the Grand Duchy of Baden. The criterion used in determining that an individual was a tramp was the absence of any fixed place of residence and only persons over 16 years old are included in the statistics. The number of cases considered was 528 (497 men, 31 women). The statistics taken related to sex, age, social condition, religion and occupation, and are exposed in a number of tables. No facts with regard to the forms of mental disease found are given and the author does not discuss any points in the psychology of this class of people.

6. *Family Murder by the Insane.*—The discussion of the case of a woman who while suffering from what the author diagnoses as puerperal melancholia, being left by her nurse for a few moments cut the throat of her six weeks old infant while not molesting the next older child which was in the room with her. He gives her family tree which shows strong hereditary predisposition to insanity. The patient had continually begged to be sent to an asylum as she feared that she would "do something" and was possessed with the fear of injuring her children. The author considers the deed as the result of an insane impulsion arising out of the painful affective condition and not due to disorientation or to delusional ideas, a causation which was long ago recognized by Krafft-Ebing in his "Legal Psychopathology."

8. *Animal Mind.*—An account of the wonderful performances of a two-year-old terrier dog belonging to a lady in Mannheim which the author was able to witness in company with Kräll, the owner and teacher of the famous educated horse in Elberfeld. The dog seems to have had a natural talent for reckoning, as his capabilities were discovered by accident while he was present at a lesson in arithmetic being given to the children of the house. His answers, somewhat in the same manner as those of the Elberfeld horses were given by beats with his paw, his mistress having trained him by means of a table in which each letter was represented by a certain number of beats, the most used letters by the smaller numbers, while a few generally used words such as yes (2), no (3), tired (4), etc., were also represented by numbers. His answers as given by the author are truly remarkable. His owner had on the previous day shown him a picture of Mr. Krall and had repeated his name carefully to the animal. She now

asked the dog "Rolf, do you know the gentleman?" A. "Yes." "Do you know his name?" A. "Yes." "Can you spell it?" A. "Yes." "Do so." A. "Krl." "Correct, but what is still wanting?" A. "l." "Certainly, but still something, the most important thing; pay attention, the name Krall" (with emphasis on the a). "Now." A. "a."

The dog was now rewarded with some scraps of meat and after a short rest was asked "How many pieces of meat are there still on the plate?" He answered promptly and correctly "8." He was next shown the picture of one of the Elberfeld horses and asked "Do you know what his name is?" A. "Yes." "What then?" A. "Zarf." "Good, there is wanting only a trifle in his name" "Zariff" (with emphasis upon the second syllable) "what?" A. "i." The dog then played with the children like any other pup. After he was rested he was exercised in picking out some coins, then the maid brought in, folded in two separate packages some numbers and their square and cube roots which she had copied from a table. Mr. Krall picking out two at random and showing them to the dog said "Pick out respectively the square root and the cube root of these numbers." The animal seemed to think for a few moments then selected correctly " $\sqrt{3,969} = 63$ and $\sqrt[3]{110,592} = 48$." (It is not so stated, but presumably he must have been exercised in the sequence of numbers before (Ref.).) After a while his mistress said to him "Listen, the gentleman is a friend of Papa's from Heidelberg and is named Wilser; have you understood?" A. "Yes." "What was the name?" A. "wim." "Hold on! You do not seem to have heard correctly. Shall I repeat the name to you again?" A. "No." "You know it then?" A. "Yes." "Then what is it?" A. "lsr." The animal had not heard the name before. Mr. Krall patted his head and asked "Shall I take any message from you to my pupils Muhamed and Zarif?" A. "Yes." "What then?" A. "Grus" (greeting). The mistress then said "That is enough for to-day. What do you say to the gentlemen who have visited you to-day?" A. "Ad." This was a stray dog picked up on the street about whose antecedents nothing could be learned. If the author is not deceived he certainly is a wonderful animal and bears out his idea that the difference between the human and the animal mind is quantitative rather than qualitative, though the distance between the two is still immense.

9. *The Development of the Building of Insane Asylums.*—A sketch of the development of institutions for the care of the insane in Baden from wards in general hospitals or in poor houses to the projected new Rastatt Asylum which is to care for 2,050 inmates and is to be constructed as follows: 1. Houses for 100-120 patients of two stories and divided into 4 wards with a central portion with rooms for offices, nurses' quarters, etc. These are to be adapted for observation and receiving wards, and for the feeble patients, the criminals, etc. 2. Similar houses but for 50-60 patients only, especially adapted for disturbed patients. 3. Double pavilions of 60-70 beds, containing on the ground floor at each end a day room, in the middle and observation ward for bed patients with 25-30 places, while the rest of the patients are to sleep in a dormitory upstairs. These buildings are intended for the antisocial, some of the disturbed and the partly disturbed who require considerable watching. 4. Simple country houses for 35-40 patients, a day room and perhaps a smaller dormitory on the first floor, the main dormitory upstairs, while the bathrooms, clothes rooms, etc., are in the basement. There is to be a villa for convalescents contain-

ing on each floor 20 beds, also a closed house of about 40 beds for patients of the better classes and an open villa of about 20 beds for the same sort of patients. There is also to be a pavilion of 30 beds for the reception and observation of mentally abnormal children; on the men's side an extra strong building of 50 beds and on each side an isolation house for infectious diseases of 30 beds. This last is to be divided into small departments, and there is provision for the separation of 15 typhoid carriers. The whole institution is to consist of 35 buildings for patients, 17 buildings for offices, shops, etc., and about 24 houses for employees, that is, of 76 buildings large and small. The pavilion system the author thinks is altogether the most desirable form for an asylum yet developed.

10. *Basedow Psychoses*.—The question whether there is any special psychosis in connection with Basedow's disease has been considerably discussed. In the last edition of his text-book Kraepelin has devoted a chapter to the subject, while a number of other authors have made contributions to it. Basedow patients are in general considered as subject to sudden changes in their affective condition, irritability and unrest while euphoria in the main predominates. The author gives here the histories of two men, the first 33 years old who since a head trauma in early life had shown psychic inferiority, restlessness, inability to settle down to any employment, tendency to complain of persecutions on the part of his family, particularly his father, whom he hated, and subject to periods of excitement on account of which he finally had to be committed. His mother had died of tuberculosis and he presented signs of slight involvement of both apices, had a struma, slight tachycardia, tremor and exaggeration of reflexes. Operation upon the thyroid was at first declined, but finally after he had remained over a year without improvement, being subject to exaggerations and remissions in his disturbed condition consent was secured and the left lobe of the thyroid was removed. He made a good recovery from the operation and his mental condition greatly improved, in that he quieted down, could sleep without hypnotics, the extreme lability of affective condition and fallacious perceptions disappeared and he gave on ordinary observation the impression of a fairly self-contained and controlled person, while psychological examination disclosed no marked defect of intelligence. He spoke respectfully of his father but explained that they took too different views of life to agree well. Still he could hardly be considered to-day as an entirely normal person but is still peculiar. While the case agrees in most respects with Kraepelin's description of a Basedow psychosis, there are too many other factors in its etiology for it to be considered as due to this alone.

The second case is that of a man, an artist, at the time of his admission 26 years old, the youngest of 9 children, one uncle insane. He developed mental symptoms when 25 years old, had been in several other institutions before he came under the author's care when 28. He showed alternating periods of excitement and quiet, with from time to time stereotypy, negativism and Ganser's symptom with fallacious perceptions and was on occasion violent and destructive. During his periods of disturbance there was swelling of the thyroid, tremor, tachycardia and exophthalmus, which all decreased as he quieted down. He was always extremely resistive, so that exact examination of him was never possible. The author says that if he had to give a diagnosis he would call the case one of dementia præcox of the hebephrenic form combined with the manic-depressive psychosis. The connection of the Basedow symptoms with his periods of excitement appears unmistakable.

11. *Forensic Importance of Chorea.*—Discussion of the case of a wagon driver who when 48 years old fell from his wagon and sustained an injury to his head, consisting of some deep scalp wounds and followed by symptoms of concussion of the brain, though there was never any evidence of fracture of the skull. From this time a gradual decrease of his mental capacity began to be more and more evident, and two years later he developed involuntary movements of the arms and legs diagnosed by competent observers, the author among others, as chorea. As there was some dispute as to what extent his capacity had become impaired and what compensation should be paid to him, he was repeatedly examined by physicians representing one side and the other who varied in their estimation of his disability and even in the diagnosis, some holding out for athetosis and gross brain injury. In 1911, six years after the accident, the patient's mental condition having further deteriorated, he made an attempt at suicide, inflicting a flesh wound of the abdominal wall upon himself with a knife and drinking 50 grm. of "Russian alcohol" (90 per cent. alcohol with turpentine, 5 per cent. ether, ammonia, and 2 per cent. Spanish pepper). This latter set up a gastritis which greatly reduced his strength and he died two weeks later of thrombosis of the bloodvessels of the lungs. The brain showed no changes beyond a flattening and some narrowing of the convolutions. In the dispute as to whether the accident was a cause of his mental condition, hence of his suicide, the author was called in and gave the opinion that the case was one of chronic progressive chorea, the mental changes being part of the disease, and that the accident could fairly be estimated to have acted as an exciting cause.

12. *Lunacy Laws in Servia.*—A résumé of the laws regulating the management of the Servian Curative and Custodial Asylum at Belgrade and the commitment of the insane. If a person is supposed to be insane a member of the family or some official must notify the police, when a physician is sent to examine him and if necessary to have him transferred to the nearest hospital. The hospital authorities must then communicate through police officials with the Minister of the Interior who designates three physicians who examine the patient together, then make out a statement which is handed to the police who then notify the Minister of the Interior. The Minister then decides whether or not the patient needs asylum treatment, and if he does, orders him committed to the first department, *i. e.*, the curative branch of the asylum. If the patient or his friends resist committal they must make a written protest to the Court which must then call upon the police for all the facts in the case, and in case the patient is unable to employ counsel must assign a lawyer to defend him. A time is then set for the hearing and the patient, his friends, such witnesses as are needed and finally the doctors are called to testify. The Court then renders a decision and orders the disposition of the patient. A person can only be declared a lunatic by judgment of the Court. An appeal from the order of the Minister of the Interior can be taken in a similar way and the Minister is then represented before the Court by the District Attorney. The Court can if it thinks necessary designate other physicians to examine and report on the case. After the patient has been in the curative part of the asylum for some time and it is decided that the case is incurable a commission of three physicians must examine him and report to the Court which then declares him insane, and only after this formality can he be transferred to the chronic or custodial department. To discharge a patient

a commission of three doctors must examine him and certify to the Minister of the Interior as to his recovery. His discharge is then ordered. The Director of the Asylum cannot discharge him on his own responsibility. The author, who is the present Director of the Asylum does not regard these laws as very modern or workable.

13. *The Deaf Mute as a Witness in Court.*—A discussion as to the admissibility and reliability of testimony given by deaf mutes à propos the trial of the Director of an institution for patients of this class who was accused of immorality and maltreatment of some of his charges. The mere fact that a person is a deaf mute does not in itself imply that his mentality is such as to render his testimony inadmissible. This must be considered as a separate question, though the fact that he suffers from such a severe defect should arouse the suspicion of injury to the other functions of the brain also, hence the general character and attainment of such a witness should be carefully considered before it is definitely decided to admit his testimony. Again, in probably the majority of cases an interpreter, usually a mute himself, who can talk in sign language is needed and his reliability and tact in translating the questions must be established. His observations in this trial showed the author that deaf mutes have certain marked peculiarities of expression, that their conception of certain ideas is quite unclear as compared to that of the average individual. For instance there was difficulty in making one of the girl witnesses understand what was meant by "unchaste" and she finally said that the wind was this, because it blew her skirts up. There is a marked difference between those completely deaf and those who have retained a rudimentary hearing and who can be taught to speak. Still the speech of these is frequently not clear to ordinary persons and may need to be interpreted by one familiar with deaf mutes.

14. *Self-Mutilation in General Paresis.*—A man 37 years old, who had been a teacher of gymnastics, became depressed following financial losses and when examined by the author presented the somatic signs of general paresis. It was recommended that he be committed to an asylum, but since his wife refused to consent to this and he had shown no tendency to injure himself and was not antisocial it was finally agreed that he should be placed in a private sanitarium for nervous diseases with the understanding that he should be at once transferred if there were any signs of trouble. He conducted himself in an orderly manner and seemed satisfied. On the evening of the fifth day of his stay he was somewhat restless and was given a mild hypnotic. At 2:30 he called the night nurse saying that he could not sleep and was given by her some medinal and watched for a half hour until he fell asleep. Next morning as everything remained unusually quiet in his room the nurse entered at 8:45 and found him collapsed in bed he having with a knife cleanly severed his right hand at the radio-carpal joint. He was at once rushed to a hospital where the stump was properly managed and recovered. He did not appear to have experienced any pain. The incident shows how suddenly the impulse to violence may come on in a paretic and how unsafe it is to leave such patients without proper watching.

15. *Epilepsy and Sedobrol.*—Those having charge of a number of epileptics having tried one after another the various medicaments recommended, usually in the end discard the others and rely upon a salt-poor diet and an amount of alkaline bromide suited to the needs of the case. A valuable addition to our therapeutic armamentarium has been found by the author

to be Sedrobol prepared by Hofmann-La Roche & Co., which is supplied in 2 gm. tablets each containing 1.1 NaBr together with certain organic constituents and which dissolved in water forms a basis for a palatable soup, to which other salt-free savory additions may be added according to the skill of the cook. He finds this preparation particularly well taken by children.

16. *Julian-Claudian Rulers.*—An interesting study of the sovereigns of this famous family as they are depicted in the writings of various historians ancient and modern. From constant intermarriage both good and bad traits became accentuated and in the later rulers the latter seem to have predominated. That the craze for power, excesses, ideas of persecution and cruelty manifested by many of them was in large measure due to the times and their situation seems undoubted and on account of the frequency of intrigues and conspiracies the fear of violent removal was in most instances only too well founded. Also since many accounts of them have come down to us from those openly hostile, their bad qualities have no doubt, in some instances at least, been exaggerated. Julius Cæsar in spite of his greatness is asserted by Suetonius to have suffered from epilepsy and to have been homosexual. Augustus is estimated by our author as mentally entirely normal, though he may have had some weaknesses of moral character. Tiberius is variously estimated and has been asserted to have suffered from attacks of melancholia, but our author thinks that though accounts of him show that he was peculiar and of a melancholic disposition he was never really insane. Caligula he considers as having suffered from congenital weakmindedness and thinks that his impulsive cruelty suggests an epileptic trait. Claudius was a degenerate, slow to develop and badly brought up, of weak will and with a tendency to sudden outbursts of rage. His degeneration can be traced to consanguinity and many of his bad qualities seem to point directly back to certain of his ancestors. Nero suffered from an excessively bad heredity especially upon the female side and can be considered as a psychopathic personality, a degenerate with contrary sexual tendencies. The author cannot find that any of the rulers of this house were afflicted with what Parsenow has described under the name of "Caesarenwahn" (feeling of near approach to the Deity, unrestrainedness, cruelty and ideas of persecution, *i. e.*, fear of being removed).

Although it is not so designated, this number of the Zeitschrift may well as considered a "Schüle Festschrift" since all the authors have been at one time or another assistants at Illeau under the directorship of this Nestor of the psychiatrists.

C. L. ALLEN (Los Angeles).

The Journal
OF
Nervous and Mental Disease

An American Monthly Journal of Neurology and Psychiatry, Founded in 1874

Original Articles

PROGRESSIVE VAGUS-GLOSSOPHARYNGEAL
PARALYSIS WITH PTOSIS. A CONTRIBU-
TION TO THE GROUP OF FAMILY
DISEASES¹

BY E. W. TAYLOR, M.D.

BOSTON,

ASSISTANT PROFESSOR OF NEUROLOGY, HARVARD MEDICAL SCHOOL; CHIEF OF
NEUROLOGICAL SERVICE, MASSACHUSETTS GENERAL HOSPITAL

With the increase of our knowledge of the so-called family diseases of the nervous system, it has become increasingly evident, whatever ultimate classification may be adopted, that these diseases may roughly be divided into two groups: First, those which, although distinctly hereditary in character, are too varied in symptomatology to permit of classification as disease entities; and secondly, those which are absolutely sharply defined in their manifestations. In the first group, may in general be included the various forms of atrophy and dystrophy of hereditary type, which run into each other in such a way as to preclude the possibility of distinct individuality. On the other hand there are certain conditions which on account of their constant manifestation may properly be regarded as entities, whatever their broader relation to the hereditary group in general may eventually be shown to be. In this group, amaurotic family idiocy, family periodic paralysis,

¹ Read at the fortieth annual meeting of the American Neurological Association, May 7, 8 and 9, 1914.

Thomsen's disease, are somewhat typical examples. The condition about to be described may properly be classed with this latter group because of the absolute definiteness of the signs and symptoms which so far as ascertainable have occurred in identical form in all the cases which have been observed. So far as I am aware, this type consisting in paralysis of the vagus-glossopharyngeal group as concerned in the act of swallowing, together with ocular ptosis, has not hitherto been described. The history of the case which has come under observation is as follows:

V. P., Figs 5 and 6, a French Canadian by birth, 59 years old, married, presented herself on November 11, 1913, at the Laryngological Department of the Massachusetts General Hospital, with the complaint that she was unable to swallow so well as formerly, that she was growing worse, and that she thought her "throat passage" was becoming smaller. She had noticed the difficulty over a period of two years. Examination in that department



FIG. 1. From this chart it appears that the parents of the generation to which the patient belongs had seventeen children, of whom eight died under the age of ten, seven lived to adult life, two males dying of the disease at the ages of sixty-seven and sixty-eight, respectively, a third male dying at forty-five of some other affection. Of the four females one has died of the disease at the age of sixty-eight, the patient under observation has the disease and is now fifty-nine years old. Another female died at the age of forty-seven of some other disease, leaving but one living at the age of fifty-seven who, so far as ascertained, has not been affected by the family disorder. It will be seen, therefore, that with but one exception, all the members of this family who have attained the age of fifty or more years have died of the disease. The matters of special interest are the undoubted similarity of the affection in all of the cases and the fact that although definitely hereditary in character, the disease has not been known to begin before the fiftieth year. The next generation numbers thirty-six, the oldest being forty-two. The disease has not appeared so far as known in any member of this generation. The next or fourth generation already numbers many children without record of the appearance of the family affection. An intensive study of this family for the next fifty to one hundred years is clearly a matter of much interest and importance.

showed that both the pharynx and the larynx were normal and that a bougie could be passed into the stomach without resistance. An X-ray threw no light on the matter, and a provisional diagnosis of cardiospasm was made. The patient was referred to the Neurological Department with a request for an explanation of the cause of the difficulty in swallowing.

Examination at this time and on many subsequent occasions gave the following facts: She first noticed a tendency to drooping of the eyelids about four years ago. Following this at an interval of about a year, she began to have difficulty in swallowing. Both of these conditions had grown gradually worse, so that at the present time (summer of 1914) she swallows with very considerable difficulty and is only able to take solid food by combining it with a large amount of water or tea. Otherwise, as she expressed it, "the food seemed to clog up in the passage." Beyond a tend-



FIG. 2. Mother. Died of the disease. Age 77. Ptosis. Paralysis of deglutition.

ency to constipation, she had no further complaints. Her bodily functions were normal and, except for the difficulty mentioned, she regarded herself as well.

She was the mother of ten children, and had had no difficulty at their birth. She had had no miscarriages, and had always led a life of activity and hard work.

Examination in detail was as follows: Cranial nerves: The first nerve showed no abnormality; the second, no impairment of vision; the fundus normal; the discs somewhat indistinct in outline, with marked physiological cupping and no swelling; the vessels of normal appearance. Third nerve, equal ptosis of both eyes, partially covering the pupils (see Fig 6). The eyes could be closed with sufficient muscular strength; the ocular movements were normal in all directions. There was possibly a slight nystagmus on the right when looking inward; the fields were normal. Pupils were moderately dilated and reacted promptly to light and

on accommodation, as well as consensually. Fourth nerve, normal. Fifth nerve, no disturbance either in the sensory or motor branches. Sixth and seventh nerves, normal. Eighth nerve, watch-tick heard at about four inches on each side; air conduction better than bone conduction. Ninth and tenth nerves, palatal reflexes well preserved; no disturbance in breathing, in phonation, or in other functions subserved by this group of nerves, excepting a pronounced difficulty in swallowing, particularly solid food. It was possible to take soft food and liquids, although on



FIG. 3. Son. Died of the disease. Age 67.

testing, the difficulty was apparent. Within the last few weeks, difficulty in swallowing had markedly increased. The eleventh nerve showed no defect. Twelfth nerve, tongue rather slender, protruded in the median line without tremor and with absolutely no evidence of atrophy. It was freely movable, and there was no speech defect whatever.

The arms showed no abnormality either in strength, sensation, coördination, or reflexes. The legs were normal in strength and without evidence of ataxia or other disturbance. Knee-jerks were normal. There was no Babinski response, no clonus, normal but slight Achilles; no objective or subjective disorder of sensation in the legs; no Romberg sign. There was no evidence of disorder of the internal organs with the exception of the circulatory apparatus and the kidneys. The heart was negative except for an accentuated second aortic sound. The pulse was 84 and full; the blood pressure on one occasion, 200 systolic; on another, 230 systolic, 115 diastolic.

Examination of the urine on April 25 was as follows: Pale; sp. gr. 1002; reaction slightly acid; albumin, slightest possible trace; no sugar. Sediment: small amount containing several epithelial cells, typical bladder and vaginal, and a few large round



FIG. 4. Son. Died of the disease. Age 68.

cells from the pelvis of the kidney or neck of the bladder. Several leukocytes; a few mucin shreds, a few uric acid crystals, and a small amount of amorphous urates.

On one occasion, the amount passed in twenty-four hours was estimated as three pints, which presumably was an error in observation. (It has not been possible to keep the patient under close hospital observation.)

Examination of the blood showed hemoglobin 95 per cent.; Reds: normal size and shape, no poikilocytes, micro- or macrocytes; no blasts; no organisms; no acromia; no stippling; no increase of platelets; no excess of fibrin. Whites: differential counts of 318 cells; polymorphonuclears (neutrophils, 72.9 per cent.; eosinophils, 1 per cent.); basophils (large mononuclears, 4.4 per cent.; small mononuclears, 19.7 per cent.); transitional forms, 2 per cent. Wassermann reaction in the blood negative.

Family History.—The patient's mother died at the age of 77, and for many years preceding her death, suffered from an increasing difficulty in swallowing together with a definite ocular ptosis. Death ultimately resulted from starvation (Fig. 2). Her father was normal so far as known. There were fifteen children of this marriage, of whom eight died under the age of ten. Of those living to adult life, there were four females and three males. Of these, two males and one female have died at the ages of 67, 68 (Figs. 3 and 4) and 68 respectively, with precisely the same



FIG. 5 Daughter. Before onset of the disease (patient under observation).

symptoms as those of the mother, and of the patient whose history is detailed above: namely, paralysis of deglutition with ptosis. One male of this generation died at the age of 45 of another affection; another female at 47, also of some other disease; leaving only a single female living excepting the patient. This woman is now 57 years old, and so far as ascertained is unaffected by the disease. It is a tradition in the family, which has been amply borne out by the facts so far observed, that the

disease never begins before the fiftieth year, a fact certainly of extreme interest, to which allusion will later be made. With one exception, all the members of this generation have been married and have had large families, for the sake of clearness omitted from the accompanying chart. Inasmuch as none of these have



FIG. 6. Same as FIG. 5. Definite signs of the disease. Age 59. Ptosis and progressive paralysis of deglutition (patient under observation).

reached the age of fifty years, according to the family tradition, the disease has not yet appeared.

From the foregoing family history, it appears that with one exception every member of the generation to which our patient belongs who has reached the age of fifty years has died of the disease. Although detailed information regarding those who have died is lacking, it has become definitely established in the family that starvation with drooping of the eyelids occurred in each case, with striking similarity. The definite character of the disturbance with its inevitable fatal outcome renders this testimony trustworthy. An unmarried sister of the patient died at the age of

68 in an institution under the charge of a Catholic sisterhood. The sister superior has kindly written me the following statement regarding this patient. She says in her letter, "This person (A. B., a sister of my patient) came to our institution some years ago, not because she was at the time very ill but rather because she was aged and wished to finish her days in our home. She suffered from paralysis of deglutition which resulted in starvation. She had a very peculiar affliction of the eyes also which I have never seen before. She died here about five years ago; our house physician attended her. He relieved her somewhat, but could not cure her." There can be no doubt that these symptoms were identical with those from which my patient is now suffering. No definite information is obtainable of the mother's illness or of the two brothers who have died, beyond the essential fact that they all starved to death and had a peculiar drooping of the lids.

I am unable to find any reference in the literature of the family hereditary affections to this disturbance. It is unique in the fact of the cleancut character of the symptoms and signs which have appeared in all the persons affected in the same form, namely: ptosis and paralysis of deglutition without other evidence of disorder of the nervous system. The fact that it never has occurred before the fiftieth year is likewise of peculiar interest. In the recently published "Handbook of Neurology" edited by Lewandowsky, Jendrassik writes an exhaustive article on the hereditary diseases, followed by a bibliography of nearly five hundred references, without allusion to the symptom-complex under discussion. Under the heading, "Hereditary ptosis and the hereditary ocular muscle paralyses," cases are mentioned in which ocular paralyses often associated with facial paralysis occur both as congenital affections and also developing in later life. Jendrassik himself has observed in two sisters at the thirtieth year a gradually developing ptosis followed by complete external ophthalmoplegia, paresis of the facial nerve, and loss of the patella reflexes. In these cases there is also a certain disorder of speech. It is hardly necessary to state that such cases bear only the most distant relation to those under consideration, owing to the widespread character of the paralysis and a coincident involvement of the spinal cord. Bernhardt,¹ in a paper entitled "*Beitrag zur Lehre von der familiären Erkrankungen des Nervensystems*," in

¹ Bernhardt, Virchow's Arch., 1891, CXXVI, 59.

1891, described certain cases, all occurring after thirty, in which there were marked bulbar symptoms associated with speech defect and spasticity of the extremities. Beyond the fact of a bulbar involvement, these cases likewise evidently bear no intimate relation to the affection under discussion. Oppenheim, in his discussion of progressive bulbar paralysis, alludes also to an apparently hereditary type occurring in children as described by Fazio, Charcot, Londe, and Brissaud-Marie, evidently a family character, in which also certain stigmata of degeneration occurred. This condition is, however, so far removed from our cases that they require no further description, if for no other reason than that it appeared in childhood.

Classification.—It is difficult to classify these cases in any recognized group. The various myopathies, ably discussed with regard to their interrelation by Oppenheim and Spiller at the last international congress of medicine in London, cannot be considered to include this type. There is, in the first place, no evidence that the disease is to be regarded as a primary muscular affection; the pathology is rather to be sought in a curiously selective process of degeneration occurring in a limited number of cells of the vagus-glossopharyngeal group, and a single cell-group of the oculomotor nucleus. It should, in this connection, be borne in mind that the eyelids contain smooth muscle fibers innervated by the sympathetic, the function of which is to widen the lid opening. The paralysis of these fibers may produce a narrowing of the space between the lids, giving rise to an incomplete ptosis. When, however, a ptosis is due to this cause, there is commonly a reduction in size of the pupils, which did not occur in this case; nor was there other evidence of sympathetic involvement. It is fair, therefore, to presume that the ptosis is not due to this unusual cause, particularly in view of the associated difficulty in swallowing.

The peculiarity of the affection lies in the fact that although very chronic in course, extending over a long period of years in each instance, no other nerves are involved, thus differentiating it sharply from an ordinary type of bulbar or pseudo-bulbar paralysis. There is no record that the hypoglossal nerve is ever affected. It certainly shows no sign whatever of degeneration in the case which I have had the opportunity to observe.

A more important consideration is the possibility that the

affection might be considered a form of myasthenia gravis. The photographs of the various persons affected lend a certain weight to this idea, inasmuch as the ptosis alone is visible, which constitutes a conspicuous characteristic of the myasthenic face. It is, however, exceedingly unusual that the weakness in myasthenia gravis is limited consistently to single muscles, as is clearly the case in the affection under consideration; nor is there any evidence to show that in myasthenia gravis the disease occurs in precisely the same form on an hereditary basis. Furthermore, myasthenia gravis usually manifests itself before the fiftieth year. It is not possible to demonstrate the myasthenic reaction, or the fatigability of the muscles beyond what one would expect in any case of muscular weakness, such as our patient presents. Furthermore, the tendency to remissions, as frequently observed in myasthenia, is wholly lacking in this case. One is, therefore, justified in excluding the symptom-complex from the ordinary myasthenia gravis; and even if further investigation should prove that it might be classified in this category of disturbances, the fact of its peculiar localization and constant appearance in hereditary form, only occurring after the fiftieth year, would still render the affection unique.

Etiology.—In the present state of our knowledge, any attempt to discover an adequate cause for the appearance of such a disorder in many members of a single family must prove futile. No doubt it may properly be considered one of the abiotrophies as described by Gowers; but this manifestly does not serve as an explanation. That there is a defective vitality may readily be assumed, but why such a defect should occur with uniformity in just these nerves remains wholly obscure. It is furthermore an added mystery that such a defect should never manifest itself until well past middle life, and in otherwise perfectly healthy persons without apparent hereditary defects or other nervous disability. The theory of over-use as propounded by Edinger likewise does not offer even a workable hypothesis. Sclerotic changes in the blood vessels occur to one as a possible explanation but although such changes may well underlie the condition, the fact of chief importance is naturally not touched, namely: that just these nerves are affected by such a process and no others. It is clearly necessary to go behind any such superficial explanation if we are to arrive at a reasonable etiology, and this is at present

impossible. In our case there is strong probability of a coincident renal affection of a degenerative sort, as shown by the urinary examination; but this fact in no way helps towards a solution of the main question.

Prognosis and Course.—My patient noticed her first symptoms about four years ago. She now has a considerable degree of ptosis, as shown in Fig. 5, but is still able to swallow, although with much difficulty, and shows no signs of emaciation from lack of nourishment. It is probable that she will live several years, so that in her case the course of the disease may be approximately estimated at seven or eight years. Beyond the fact that the other members of the family dying of the disease are known to have been ill for a long period, no definite data are at hand. In this respect the degeneration does not differ from others involving cranial nerves important to life.

Summary and Conclusions.—A clearly defined disease or symptom-complex exists not hitherto described, consisting of ocular ptosis and paralysis of deglutition coming on after the fiftieth year, and leading to death without further involvement of the nervous system.

The affection is certainly to be classed among the family hereditary diseases, as shown by the fact that it has occurred in two generations, in the second affecting all but one of those who reached the age of fifty years.

The disorder is peculiarly remarkable in that although hereditary in type, it has not been known to occur in early life, and must therefore be regarded as superinduced in predisposed persons by conditions arising in the degenerative period of life.

The justification for regarding the affection as a distinct entity lies in the facts that so far as ascertainable it occurs in precisely the same form in all persons affected and also invariably in the declining years of life.

PUNCTURE OF THE CORPUS CALLOSUM WITH SPECIAL REFERENCE TO ITS VALUE AS A DE-COMPRESSIVE MEASURE¹

BY CHARLES A. ELSBERG, M.D.,

NEW YORK

In order to institute the proper treatment for the relief of conditions of increased intracranial pressure, the neurologist and the surgeon must have a clear understanding of the physical changes that occur in the pressure conditions within the skull cavity from a growing tumor or expanding lesion. We are apt to think that the encroachment of the growing tumor is the main cause. The increase of pressure is due not only to the added bulk of the new growth, to the cerebral edema which accompanies intracranial neoplasms and first described by Reichardt, or to the hyperplasia of the affected lobe recently demonstrated by Spiller, but also to the internal hydrocephalus which so often complicates tumors of the brain.

The free communication between the ventricles and the subdural spaces of the brain and cord is a necessity for the proper functionizing of the brain; when the cerebrospinal fluid can no longer flow from the lateral ventricle through the Sylvian aqueduct to the fourth ventricle and the spinal canal, symptoms due to increased intracranial pressure soon appear, which are well known and which the surgeon has attempted to relieve in a variety of ways.

The general symptoms of increased intracranial pressure are therefore often due to the stagnation of the cerebrospinal fluid and the distension of the ventricles. The same condition may result from an oversecretion of ventricular fluid. Increase of pressure within the cranial chambers results in the compression of the yielding walls of the cerebral veins, with a resulting congestion and edema, and the collection of edematous fluid in the ventricles. This is especially apt to occur when a tumor grows

¹Read at the fortieth annual meeting of the American Neurological Association, May 7, 8 and 9, 1914.

in a location where it will exert direct pressure upon the veins in the interior of the brain, upon the choroid plexuses and the veins of Galen. Marked distension of the ventricles occurs early in tumors in the posterior fossa where pressure upon the aqueduct is frequent.

A careful investigation of a large series of brain tumors has shown me that more or less distension of the ventricles is present in about seventy-five per cent. of cases in which a new growth is found at operation or autopsy (372 out of 500 cases); the intraventricular fluid must be a very important factor in the causation of the increase of intracranial pressure in these cases.

Is it not surprising that in the cases in which the cause of the raised pressure could not be removed (*i. e.*, a tumor excised, cyst or abscess drained, etc.) so much attention has been paid to the methods for diminishing the tension by the removal of part of the bony wall of the skull and the incision of the dura (decompressive craniotomy), and so little to means for the permanent decrease of the important ventricular distension? Repeated lumbar puncture has sometimes been performed with this purpose in view, but it is only a temporary measure, and harmful or even dangerous in subtentorial disease.

As a palliative operation, cerebral decompression has found extensive usage in states of acute intracranial pressure elevation (fracture of the skull, etc.); in tumors of the brain there may be little or no benefit from the operation. In some instances (mid-brain lesions) a subtemporal decompression may exaggerate the symptoms. Finally, the great disadvantage of a decompressive craniotomy is that the patient is left with a deformity which is marked in proportion to the amount of increase of intracranial pressure.

Attention was directed to the significance of the internal hydrocephalus which accompanies brain tumors when a method was devised to treat an entirely different condition, congenital or acquired chronic internal hydrocephalus. Many attempts had been made to benefit these patients; the fluid from the ventricles had been drained into the subcutaneous tissues, but cerebrospinal fluid is not absorbed from the cutaneous tissue and large collections of fluid resulted. Attempts have been made to drain the ventricular fluid into the subdural spaces by means of small silver canulæ. One operator, and he a brain surgeon of wide experience, actu-

ally proposed and tried to drain the fluid into the peritoneal cavity; while another made a connection between the ventricles and one of the large veins of the neck or one of the venous sinuses in order to drain the fluid directly into the blood stream. It remained for Anton and von Brahmman to devise a simple method for the making of a permanent communication between the ventricles and the subdural space over the cerebral convexities, by means of puncture of the corpus callosum.

A small incision is made at right angles to the median line beginning one or two centimeters away from the median line and one to two centimeters behind the coronary suture. With a trephine, a button of bone is removed and a small incision made in the dura. A small bent canula of German silver is now intro-

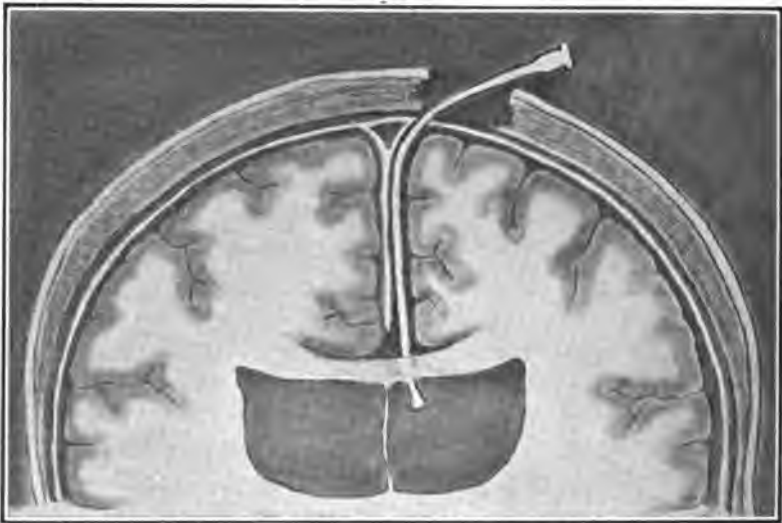


FIG. 1. The Surgical Anatomy of Puncture of the Corpus Callosum. The curved canula is seen passed through the opening in the skull and dura, by the side of the falx cerebri, and through the corpus callosum into a lateral ventricle.

duced through the small slit in the dura and pushed toward the median line until the resistance of the falx cerebri is felt. The blunt point of the canula is now slid along the side of the falx until the corpus callosum is reached. With slight pressure the corpus callosum is perforated near the genu and fluid escapes from the ventricle. (See figure 1.). The opening in the corpus

callosum is now enlarged by manipulating the canula so that a slit about one centimeter in length is made. The canula is now withdrawn, the small opening in the dura closed by one fine silk suture, and the skin wound closed. On account of the higher pressure in the ventricles than in the subdural space, fluid continually passes from the ventricles to the subdural space over the convexity of the hemispheres and the opening in the corpus callosum therefore remains patent.

The results obtained by Anton and von Brahmman in the treatment of internal hydrocephalus were very promising and led them to believe that puncture of the corpus callosum was a very satisfactory method for the relief of internal hydrocephalus if the patients were operated upon before the convolutions had become flattened and destroyed by the pressure and before optic atrophy had occurred. Of 17 cases of congenital hydrocephalus operated upon by von Brahmman, the optic neuritis had gone on to almost complete atrophy in 15 of them before the patients came under observation. Spasticity and ataxia were diminished even in these patients, the intelligence was improved in many, and some of the children developed normally after the little operation. They then tried the operation in other cerebral conditions. Up to the time when their monograph was published they had operated upon 17 cases of simple hydrocephalus, 5 of tumor of the hypophysis with dilatation of the third ventricle, 23 cases of tumor in or near the ventricles, 4 cases of epilepsy, 2 of non-suppurative meningitis, and 1 of "Turmschaedel." A number of the patients were much improved after the callosal puncture.

During the past two years I have performed puncture of the corpus callosum 37 times. The operation has never been difficult and all of the patients recovered from the little operation without any bad after effects.

PUNCTURE OF THE CORPUS CALLOSUM IN THE NON-OBSTRUCTIVE HYDROCEPHALUS OF CHILDREN

I have operated upon 7 children who suffered from the non-obstructive variety of hydrocephalus. All had much enlarged heads, with more or less spastic extremities and with the retinoscopic evidences of different grades of optic atrophy. Two of the patients were much improved. In one child in whom callosal

puncture was done about three months ago, a very marked improvement in all of the symptoms occurred. The child was apparently blind, the pupils did not react to light, there was marked spasticity of all of the extremities, marked nystagmus, and great enlargement of the head. Two months after the operation, the head measured five centimeters less in all directions, the pupils reacted well to light, the child seemed to recognize objects about it, and nystagmus and spasticity had almost entirely disappeared.

From the reports in the literature and from my own experience I believe that puncture of the corpus callosum is the best method up to the present time for the relief of non-obstructive internal hydrocephalus, and if the little patients come to us early enough before irreparable damage has been done to the brain and irremediable changes have occurred, we should be able to relieve entirely a great many of them.

PUNCTURE OF THE CORPUS CALLOSUM AS A DECOMPRESSIVE METHOD IN TUMORS OF THE BRAIN

A consideration of the mechanical factors involved in cerebral decompression, and the physical changes that occur when a de-



FIG. 2. Transverse Section through the Brain at the Genu of the Corpus Callosum in a case of brain tumor with marked internal hydrocephalus. A subtemporal decompression had been done. Note the marked distortion of the brain and ventricle. (From Anton and v. Brahmman, "Der Balkenstich," Berlin, 1913.)

compressive craniotomy has been done, has led me to suggest to you a viewpoint which may be somewhat novel. It seems to me

that up to the present time we have paid insufficient attention to the lines of force along which the brain tissues are being dislocated after a decompressive operation. The best decompression must be one in which the brain tissue is dragged upon or pushed in such a way that the smallest number of nerve tracts are interfered with. I have examined the brains of a number of patients

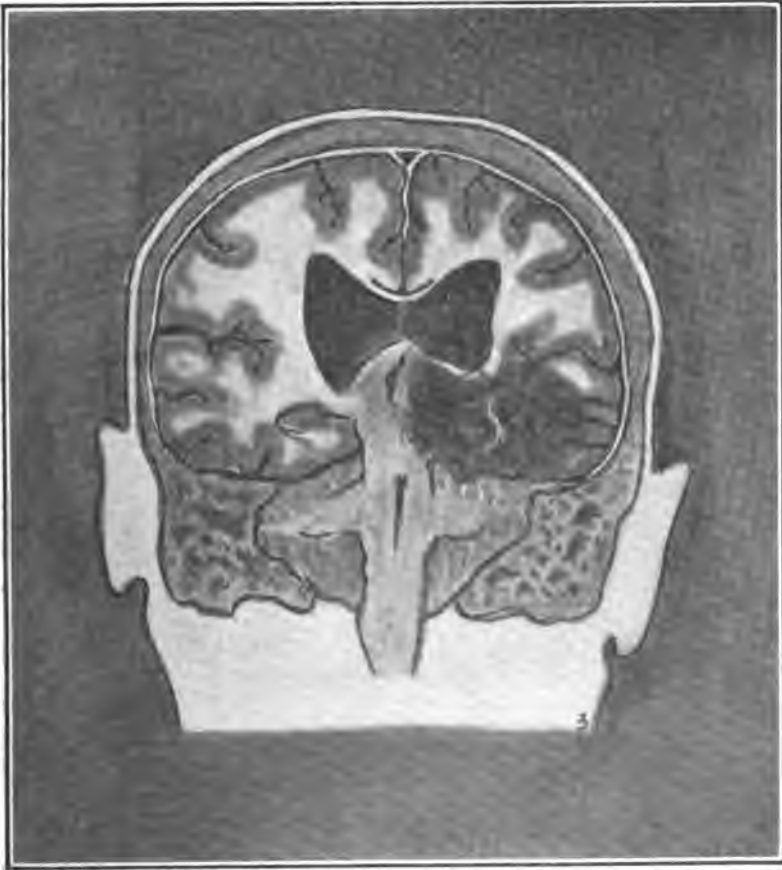


FIG. 3. Tumor of the Right Temporal Lobe with internal Hydrocephalus (diagrammatic reconstruction from anatomical specimen). In a case of this kind, the distortion shown in Fig. 2 would be apt to occur.

who have died from irremovable brain tumor and in whom a palliative subtemporal or suboccipital decompression had been done. In supratentorial lesions there was usually considerable internal

hydrocephalus, and the distended lateral ventricle of the operated side or the third ventricle was dragged upon or distorted (Fig. 2). Much harm was surely done to important nerve tracts by this unnecessary distortion of the brain, and just as much space

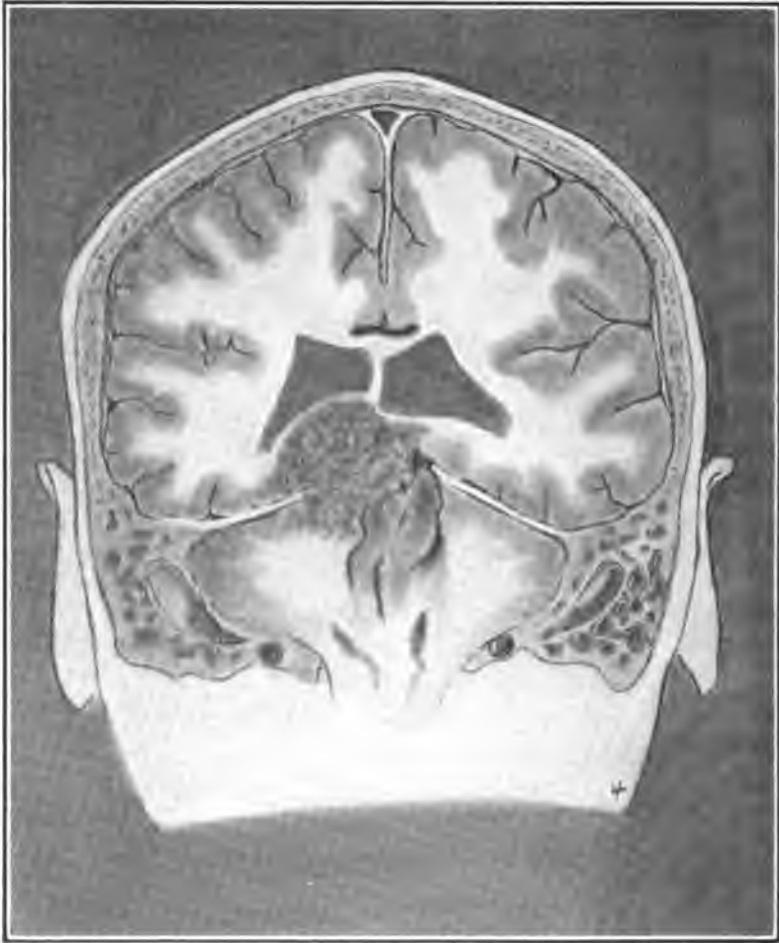


FIG. 4. Mid-brain Tumor with Hydrocephalus. A type of growth in which the symptoms would be apt to be made worse by a subtemporal decompression on the right side.

would often have been gained and mechanically a more correct decompression have been accomplished by means of puncture of the corpus callosum and permanent autodrainage of the ventricles,

perhaps combined with a decompressive craniotomy (Figs. 3 and 4.)

Motor tract symptoms are to be made worse in a left or right mid brain tumor by a subtemporal decompression on the right side; the symptoms of a tumor which lies medianward to the internal capsule may be exaggerated by a subtemporal decompression (Fig. 5). Inasmuch as a considerable amount of ven-

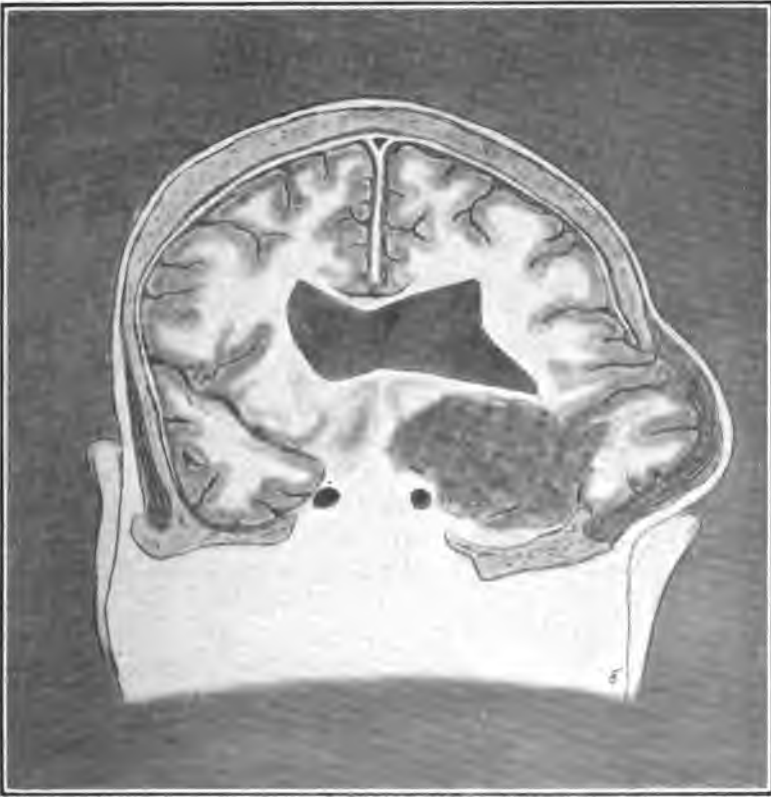


FIG. 5. Infiltrating Tumor of the Temporal Lobe with Hydrocephalus in which a subtemporal decompression had been done. In a case of this kind a puncture of the corpus callosum should also have been done (diagrammatic sketch from anatomical specimen).

tricular distension always accompanies these new growths, it would be much more reasonable to attempt to relieve the symptoms by a method which will permanently relieve the internal hydrocephalus than by a unilateral or bilateral decompression.

Similarly, much of the headache, vomiting, and optic neuritis which result from expanding lesions in the subtentorial region, are due to the high grade of internal hydrocephalus. When a palliative operation is to be done, it is therefore better to get rid of the ventricular distension by a simple operation, than to do the more serious suboccipital decompressive craniotomy.

In addition to the advantages of puncture of the corpus callosum above enumerated, the absence of a deforming hernia in the subtemporal or suboccipital regions is an advantage not to be underestimated.

In short, I believe that puncture of the corpus callosum is of great value, not only as a temporary palliative method in unlocalizable tumors of the brain, but in many instances, the best palliative operation in irremovable cerebral and cerebellar neoplasms.

I have used this method 30 times instead of or combined with decompressive craniotomies, and in a considerable number of patients have seen great improvement follow the operation. In more than one half of the patients, an immediate improvement occurred; the headache was relieved, the swelling of the optic nerve heads decreased and sometimes subsided entirely. In several cases, the operation was done without any anesthesia because the patients were in stupor or coma. In two of these, the patients became conscious and answered questions before they left the operating table. I have had one patient with advanced symptoms of a mid brain tumor so much relieved for more than six months, that he was able to return to his work. The headache and swelling of the discs disappeared rapidly, and ocular palsies subsided.

The following are examples of the results that I have obtained:

Madeline W., 3 years of age, inoperable cerebellar tumor involving the worm and both hemispheres. Puncture of the corpus callosum at the New York Neurological Institute. Papilledema before operation, 4 D. in each eye; after operation, papilledema entirely disappeared in three weeks. Drowsiness which existed before operation entirely disappeared.

Walter D., Neurological Institute, service of Dr. Bailey. Mid brain tumor, marked drowsiness, incontinence, double Babinski, papilledema 1 D. Puncture of corpus callosum. Result: Disappearance of severe headaches, normal sensorium, full control of bladder and rectum, disappearance of pathological reflexes, subsidence of the papilledema.

Dr. R., mid brain tumor or growth near a ventricle. Private patient of Dr. Bailey at the New York Neurological Institute. Increasing symptoms with advancing optic neuritis. Patient in coma when puncture of corpus callosum was done. Ventricles contained greenish colored fluid. Operation done without anesthesia. Fifteen minutes after the operation, patient was awake and conversed freely with nurses and physicians.

Jack F., 27 years of age. Service of Dr. Lilienthal, at Mount Sinai Hospital. Marked mid brain symptoms with choking of discs, ocular palsies, diplopia, very severe headaches so that patient cried out with pain if he made the slightest movement. Puncture of corpus callosum. Within two weeks, disappearance of all of the symptoms above mentioned. Patient much improved for six months.

In almost all of the patients some improvement occurred. In the patients in whom no increase of ventricular fluid was found, there was of course no change in their condition, and other decompressive methods had to be resorted to.

The operation of puncture of the corpus callosum has given us such satisfactory results that it is the operation of choice in mid brain tumors, in unlocalized intracranial growths, in subtentorial neoplasms in which a palliative operation is to be done. If the headache and the optic neuritis require a decompressive operation before the tumor can be localized, I now do a callosal puncture, and only add a subtemporal or suboccipital decompressive craniotomy either if little or no distension of the ventricles exists, or if the callosal puncture has not relieved the papilledema and the other general tumor symptoms.

The operation is only a palliative or temporizing measure, and the radical removal of the tumor should always be done if that be possible. As a palliative method, however, I can highly recommend this little operation, which deserves a more extended use than it has up to the present time received.

My experience is not large enough to state exact percentage results, nor is it possible to say how often the opening in the corpus callosum will remain patent.

PULMONARY COMPLICATIONS OF APOPLEXY¹

BY PHILIP COOMBS KNAPP, A.M., M.D.,

VISITING PHYSICIAN FOR DISEASES OF THE NERVOUS SYSTEM, BOSTON CITY
HOSPITAL

When a patient is stricken with apoplexy, the presence of râles in the lower part of the lungs is of grave omen. They not only serve to some degree as an index of the gravity of the seizure, but they indicate a new danger. Patients do not necessarily die on account of the pulmonary complications of apoplexy, but these complications often contribute to the fatal issue, and, in some cases, could they be prevented, the patient might recover from the shock.

In most cases these complications are due to one of two pathological processes,—hypostatic pneumonia, due to a more or less rapidly developing edema, or inhalation pneumonia, due to the entrance of mucus, food or germs into the bronchi from the mouth. The former process may depend upon some vaso-motor disturbance, and sometimes comes on very soon after the shock, developing with great rapidity until both lungs are filled with fluid. The accumulation of fluid follows the ordinary laws of gravitation and is first noticed in the lowest parts of the lung. Inhalation pneumonia is of slower development, coming on with the increasing invasion of the lung by foreign substances, in stuporous patients who can neither cough nor swallow satisfactorily. The two processes may occur in the same patient, and all sorts of variations in their development may be found. A patient may succumb in a few hours with both lungs filled with râles, or he may linger for several weeks, or even a month or two. Other forms of pulmonary disease are merely coincidences, but these are dependent directly upon the apoplectic condition.

The location of the lesion in the brain has little to do with the development of these forms of pneumonia. Charcot once held that the lesion caused vaso-motor changes on the opposite side of

¹ Read at the fortieth annual meeting of the American Neurological Association, May 7, 8 and 9, 1914.

the body, leading to edema, etc., in the opposite lung. Strümpell thought that the process began in the lung of the paralyzed side, owing to the diminished respiration in that lung from paralysis of the respiratory muscles. That these muscles are usually affected is an accepted fact, but the symptoms, even at their onset, are usually bilateral. In but three cases out of one hundred and twenty, did I find one lung affected, and in only one of these cases was it affected on the paralyzed side. In a fourth case of left hemiplegia râles were found in the right upper and the left lower lobes. In two cases of croupous pneumonia, the lung was affected in one case on the sound side and in the other on the paralyzed side.

My data in regard to the pulmonary complications of apoplexy are derived from two hundred and six cases in my service at the Boston City Hospital. Of these one hundred and twenty patients died in the hospital. I have not attempted in this enumeration, to differentiate between the various forms of lesion,—hemorrhage, thrombosis, embolism,—which gave rise to the apoplexy. Seventy of these cases presented the ordinary symptoms of hypostatic or inhalation pneumonia,—difficulty in breathing, slight fever, râles in the lower posterior portions of the lungs, and eventually signs of consolidation. Eight others showed slighter signs of respiratory disturbance,—harsh or irregular respiration, prolonged expiration, and the like, without râles or definite signs of consolidation. Two-thirds of the fatal cases, therefore, showed some evidence of pulmonary involvement. Out of eighty-six cases discharged from the hospital, forty-three or exactly one half, showed some disturbances, but in only sixteen were they marked. None showed the rapid filling up of the lungs often met with in severe cases, which is probably to be regarded as an agonal symptom.

I have brought up this subject not because it is particularly novel or unknown, but because the majority of the text-books on internal medicine and nervous diseases deal with it in a very inadequate manner, both in calling attention to the existence of the complications, and in regard to their treatment.

These forms of pneumonia, as I have said, are secondary to the apoplectic state, the result either of the vaso-motor disturbance arising from the cerebral lesion or the cardio-vascular disease which causes the apoplexy, or of the stupor which permits the

invasion of the bronchi and lungs by foreign substances and micro-organisms. They would not develop without the apoplexy, and apoplexy of course may cause death without any pulmonary edema or broncho-pneumonia. Nevertheless some cases might recover from the apoplexy, did not the broncho-pneumonia add to the danger.

Such being the case, one of the chief aims in the treatment of the apoplectic state should be to prevent the development of broncho-pneumonia. Various factors combine to produce the condition, all of which must be considered in the treatment. The circulation is impaired, causing stasis in the lungs, the fluid, by the action of gravitation, seeking the dependent portions. By reason of the shock and of the paralysis, the movements of respiration are lessened, so that the fluid is not forced out from the lungs. On account of the stupor, there is lessened reflex sensibility of the pharynx and larynx, so that the patient rarely coughs. Mucus, food, and the normal microorganisms of the mouth, collect in the throat and are gradually carried down into the lungs. The need of air from the diminished movements of respiration, causes the stuporous patient to breathe through the mouth, thus affording ingress to the organisms floating in the air. All these factors act together to promote the development of broncho-pneumonia in which hypostasis and inhalation play a part in varying degrees.

To combat these various pathogenetic factors, therefore, therapeutic measures should be undertaken with four different objects in view: oral asepsis, stimulation of the circulation, stimulation of respiration, and counteracting the action of gravity.

The necessity for oral asepsis admits of no dispute. Care in the administration of food, frequent and thorough swabbing out of the mouth with mild antiseptic solutions and the use of antiseptic douches or spray in the nostrils are all of advantage, although complete asepsis can hardly be attained.

Stimulation of the circulation by drugs is a measure attended with some danger and has seldom seemed to me very efficacious. Over-stimulation may increase the hemorrhage in the brain in the early stages of apoplexy, especially if drugs be given which tend to increase the blood pressure. Digitalis, ammonia, camphor and the like, given with drugs that lower the blood pressure, do no special harm, and sometimes seem to aid.

Stimulation of the respiration by general hydrotherapeutic measures is often not feasible, but the production of sudden, deep inspirations by the application of ice to the back or the epigastrium, or at times by cold douches, is sometimes helpful.

In this connection a word should be said of the very great benefit which may sometimes be obtained by venesection, where the patient has labored respiration, a full pulse, high blood pressure and marked pulmonary engorgement. The removal of eight or ten ounces of blood from the arm occasionally has a most dramatic effect in relieving the symptoms, even though it may not prevent the fatal issue.

One of the important indications for the prevention or alleviation of pulmonary disturbances is to counteract the effect of gravity by frequent changes of position. There are still many who urge that after an apoplectic shock, absolute rest should be enforced, arguing that even turning the patient in bed may cause an increase in the hemorrhage, and recommending that he be not removed from the place where the shock occurred, but be put in a bed made up on the floor. Others, recognizing the danger of pulmonary trouble, advise frequent changes of position from the first. I have seen many patients flat on their backs in bed, the head scarcely raised from the mattress by a thin pillow, and sometimes even lower than the body, the mouth open, the respiration stertorous and labored, the nurse scarcely daring to disturb him to swab out the mouth, and certainly not daring to lift the head enough to give drink or medicine. Such a condition is most favorable for the production of inhalation pneumonia and should never be permitted.

The danger of causing an increase in the hemorrhage by changing the patient's position seems to be about as great as the danger of causing delirium tremens in a case of marked alcoholic stupor by cutting off alcohol. It is my custom to have the upper part of the body raised up in bed, with the head fairly high; to turn the patient from side to side frequently, and, when the lung conditions seem threatening, to get the patient out of bed in a day or two into a chair for a short time, at least once each day, fastening him into the chair if need be. If there be a hemorrhage still going on, the ordinary laws of physics should convince us that less blood will flow out into the brain when the head is higher than the body, than when the head is nearly on the same level. I have

seen no harm from such treatment. Many patients die with some signs of increasing coma, but they show no change as a consequence of moving. Other cases may show improvement, like that often seen in getting a patient stuporous from a fractured skull or an alcoholic "wet brain" up in a chair.

Out of the one hundred and twenty fatal cases cited, four succumbed to a second shock while in the hospital. In three cases, however, the second shock did not come soon after the first, while the patient was being moved in bed or taken out of bed and put in a chair. It came several weeks later, when the patient had recovered from the first shock and the hemiplegia had improved so much that he could move about. Then, when making some slight voluntary effort, the second and fatal shock came. Some cases, too, who at first show no signs of broncho-pneumonia, may regain consciousness and improve. In a few days they gradually grow more stuporous and die with evidences of trouble in the lungs. The cause of this change for the worse is not always clear. Some have been kept quiet and not subjected to any special change of position that would bring on a fresh hemorrhage, nor has an autopsy shown any new focus of disease to explain the increase of symptoms. At all events the change in position cannot be held responsible.

I do not claim that broncho-pneumonia can always be prevented by the various methods of treatment mentioned above. The presence of râles is of grave significance, but their occurrence is merely a consequence of a still graver affection, the shock itself. These methods of treatment often fail to relieve the condition. In some cases, however, the patient might recover, but for the added burden of pulmonary complications, and, if treatment can pull even a few of such patients through, it should not be neglected. The methods are neither new nor original. Various men in this country and abroad have advocated them. Many of us, probably, have repeatedly put them in practice. In view, however, of the slight attention paid to the whole matter in many of our text-books, and the insistence upon absolute rest and the failure fully to consider the treatment by many authors, it has seemed to me worth while to call attention once more to the frequency of these complications and the best methods for their prevention.

Society Proceedings

THE PHILADELPHIA NEUROLOGICAL SOCIETY

OCTOBER 23, 1914

The President, DR. CHARLES K. MILLS, in the Chair

Dr. W. B. Cadwalader presented a case of facial hemiatrophy greatly improved by the administration of thyroid extract.

Dr. F. X. Dercum said that it was of interest to recall that thyroid extract is often of value in scleroderma and Dr. Cadwalader's case was initially a scleroderma, or morphea. There can be no doubt that diffuse scleroderma and facial hemiatrophy are closely related diseases.

Dr. Alfred Gordon asked Dr. Cadwalader whether he had found changes in the electrical reactions in his case, and, if so, what the electrical reaction consisted of, and what was the degree of improvement that followed the treatment with thyroid.

Dr. Augustus A. Eshner said he would venture to anticipate what Dr. Cadwalader might say in reply to Dr. Gordon by stating that he had seen the patient a good many times at Dr. Mitchell's clinic in the Orthopedic Hospital, and that there was no question as to distinct improvement. When the patient first came the left side of the face was smooth and the skin parchment-like in appearance. Under treatment with thyroid extract, carefully controlled, the skin has become softer and the face more mobile. There has been no loss in weight. The atrophy of the face, naturally, still persists. The patient herself is aware of the improvement that has taken place and she agrees subjectively with the impression that she had gained.

Dr. Cadwalader said in reply to Dr. Gordon's question, that there was a quantitative diminution of the electrical reactions—but reaction of degeneration was not present. Dr. Eshner had already answered the question as to the amount of improvement which had taken place. There was no doubt that the patient had improved, but she is by no means well, though decidedly better than before treatment had been undertaken.

Dr. T. Maxwell Langdon presented a patient with a peculiar form of hippus in tabes.

Dr. Tom A. Williams said that he had a case of tabes with partial optic atrophy some three years ago and gave salvarsan without any detrimental effect to the optic nerve, however, without much effect on the patient. The vision was not so defective as in the patient shown, but the man also had dilated pupils and a hippus. There was a distinct hippus without complete optic atrophy.

Dr. Spiller said he had not seen a pronounced hippus as a sign of tabes as in the case Dr. Langdon had shown. He had often seen contraction of the iris with rapid dilatation again. Fry, of St. Louis, read a paper before the American Neurological Association a few years ago

in which he spoke of the rebounding pupil. That may be a sign of tabes, and may precede the Argyll-Robertson pupil.

Dr. C. W. Burr presented a case of multiple neuritis with marked hysterical ataxia.

Dr. Tom A. Williams said Dr. Burr's case was a good illustration of Babinski's contention that suggestion which leads to hysteria is most commonly provoked by some physical disease. This man was a beautiful example of a paralysis followed by an imaginary one of a different type.

In discussing Dr. Burr's patient, Dr. D. J. McCarthy stated that he would add a correction of the notes of the case. His recollection was to the effect that the man had been admitted to his wards during the summer, that the knee jerks were lost on the entrance of the patient to the nervous ward. The case was unquestionably one of multiple neuritis. The arm reflexes were retained, but later the neuritis extended to the upper extremities with loss of reflexes in the upper extremities. The patient was in bed, in a helpless condition with paralysis of both lower extremities at the time.

A CASE OF FRIEDREICH'S ATAXIA

By James Hendrie Lloyd, M.D.

Dr. Lloyd showed a patient with Friedreich's ataxia. The patient was a young man whose chief symptoms were ataxia and a characteristic deformity of the feet—a form of pes equino-varus, with over-extension of the great toe, very similar to that shown in the picture originally published by Brissaud. The knee-jerks were abolished, and the Romberg symptom was well marked. There were, however, no Argyll-Robertson pupils, no sensory disturbances, no eye-ground changes, and no fulgurant pains. On the other hand, there was no nystagmus or speech defect—which, of course, was somewhat against the diagnosis of Friedreich's disease. In this connection Dr. Lloyd spoke of the possibility of atypical forms. The Wassermann test was negative, as were also the other tests on the cerebro-spinal fluid. The case was of additional interest from the fact that it had been reported by an advertising charlatan as a cured case of genuine locomotor ataxia.

In reference to the case presented by Dr. Lloyd, Dr. Spiller said it occurred to him while Dr. Lloyd was presenting the patient that there was another diagnosis possible, and the case might be one of the Charcot-Marie-Tooth type. The man had very small wrists comparable with the upper limbs, and small ankles comparable with the legs and thighs. He had no tendon reflexes.

Dr. Lloyd, in speaking of his case, said he believed the only suggestion made was the one by Dr. Spiller, who thought the case might be referred to the Charcot-Marie-Tooth type. Dr. Lloyd thought the objection to that was that the man has no muscular atrophy. In the Charcot-Marie-Tooth type we have really a form of peripheral multiple neuritis, or progressive multiple neural degeneration. There is not only muscular atrophy in such cases, but there may be fibrillation, and the extensor muscles are especially affected, so that the patient has foot-drop and the so-called turkey gobbler gait. This boy has not got this. Dr. Lloyd called attention to the fact that the patient has no nystagmus and no speech defect, which was against the diagnosis of

Friedreich's ataxia. It is not a familial case. One of the best cases at Blockley, however, is in a man whose history is not familial.

EXTRAPYRAMIDAL HEMIPLEGIA

By Alfred Gordon, M.D.

The patient, male, 25 years old, had at the age of eight an attack of left hemiplegia with aphasia. The aphasia disappeared at the end of several weeks. A year later the following peculiarity was noticed. Gradually a contracture developed in the wrist and foot, and a tendency of the arm to become everted at each movement was noticed. At present the patient shows the following condition:

There is a certain weakness in the left arm and leg, but no actual paralysis. He can voluntarily move every joint of the affected limbs with the exception of the wrist and ankle. On passive movements no resistance is felt, and at times hypotonia is noticeable. On motion an extreme contracture of the fingers and toes, wrist and ankle makes its appearance. Besides, the arm becomes then everted. At the same time an associated contracture is observed in the muscles of the neck, face and ear on the left side. The knee-jerks are exaggerated on both sides; there is no ankle-clonus and no toe phenomenon by any of the well known methods. No sensory disturbances. Bladder and rectum are normal. No visceral involvement. Mentality is normal. The face shows an atrophic condition on the left and its musculus frontalis is paretic. The eyes are normal. Urinalysis, blood examination and Wassermann test are all negative. Dr. Gordon discussed the differential diagnosis between this form and the classical organic hemiplegia, and also compared it with hysteria, dysbasia musculorum deformans and Wilson's lenticular degeneration. After demonstrating the differential features he arrived at the conclusion that it is a case of extrapyramidal hemiplegia, a new clinical entity. Its essential features are: Unilaterality; no actual paralysis, but a certain amount of disability due largely to the contracture; increased knee-jerk, but no toe phenomenon; preservation of voluntary movements; extreme contracture upon the least passive or voluntary movement; preservation of abdominal and cremasteric reflexes on the affected side; inclination of the trunk towards the affected side when walking; atrophic condition of the entire affected side including the face; clonic contractions of the muscles of the face on the affected side; preservation of sensation.

Dr. Spiller said Dr. Gordon stated that the man's paralysis came on in typhoid fever. That is not unknown. Dr. Spiller had had such cases, and Dr. E. M. Williams reported three such cases under Dr. Spiller's observation. The lesion usually is thrombosis.

The case presented by Dr. Gordon Dr. Spiller regarded as belonging to von Bechterew's postapoplectic hemihypertonia. Dr. Spiller had published a paper on this subject with the description of a case in the *Philadelphia Medical Journal*, December, 1899. He believed Dr. McCarty also had reported a case.

Dr. Tom A. Williams stated that the attitude of Dr. Gordon's patient resembled that seen in athetosis if the movement were absent. When it is remembered that athetosis occurs only as the result of prenatal or natal lesions, an explanation of this patient's syndrome suggested

itself in the comparison of extrapyramidal paralysis in the paraplegia of the aged, with or without pseudo-bulbar symptoms, for in them there are never athetoid movements, whereas in the extrapyramidal paralyses occurring before birth, athetosis is common. As the onset in this case was at the age of eight, we find in addition the "marche aux petits pas" and timorous walk of the aged, the attitude of the wrists such as occurs in infantile cases, but without the movements they show.

Dr. Mills said that Dr. Gordon's case was not one of ordinary typhoidal hemiplegia, but rather one of hyperhemiplegia. Dr. Gordon's opinion regarding the site of the lesion and the character of the case seemed to be correct in the light of recent views regarding the functions of the lenticula. Dr. Mills said that it was well-known to the members of the society that he had presented a paper on emotional expression, muscle tonicity and the cerebral tonectic apparatus at the meeting of the American Neurological Association in May, which paper should have appeared before this in the *Neurologisches Centralblatt*, but it had been held up for some time by circumstances concerning the war in Europe. He would only recall that in his opinion the cerebral tonectic apparatus was extrapyramidal and quite separate from the pyramidal system although intimately connected with the latter by cortical and by strio-rubro-thalamic tracts. Tone rendezvoused in the cortex of this extrapyramidal tonectic apparatus was, so to speak, delivered upon the motor (pyramidal) tracts and it was in this way that muscle tonicity could be accounted for best. Atonia, hypotonicity, hypertonicity and irregular tonicity all might result from lesions of the pyramidal tracts, but this was because of the effects which lesions of this kind had on the reception and delivery of tone and impulses from the cerebral tonectic apparatus.

Dr. Alfred Gordon said he was very glad to hear the remarks of Dr. Spiller and Dr. Mills and Dr. Williams. The hemihypertonia mentioned by Dr. Mills was, of course, present in the man. That was one of the symptoms of the condition the patient presented. As Dr. Mills mentioned, it is the absence of the toe phenomenon which would point to an extrapyramidal lesion. To Dr. Gordon's knowledge no autopsies have been as yet performed in cases of that kind. He would by analogy with Wilson's disease locate it in the lenticular region. Dr. Gordon said he would like to get information with reference to the involvement of the upper portion on the left side of the face, as there is a paresis of the frontal muscle. He believed that we are justified in considering this condition as an extrapyramidal hemiplegia. While it is not a true hemiplegia, nevertheless cases of that kind could be called extrapyramidal hemiplegia. The case was interesting to him because it was quite novel and goes hand in hand with Wilson's disease, except only that it is only on one side. Dr. Gordon said that perhaps he had given the wrong impression in reference to the occurrence of organic hemiplegia in typhoid fever. Several years ago he exhibited before the County Medical Society cases of hemiplegia occurring in typhoid fever. He did not mean to intimate that they are rare, although they are not frequent.

Dr. William Drayton and Dr. T. Maxwell Langdon presented a case with lost tendon reflexes and Argyll-Robertson pupils and no other signs of tabes.

Dr. F. X. Dercum and Dr. Willis F. Manges presented an interesting case of brain tumor; localization by means of x-rays.

Dr. D. J. McCarthy in discussing Dr. Dercum's paper wished to place on record a man with syphilis and multiple sclerosis where the x-ray picture showed, apart from the multiple sclerosis, a lesion very much like that in Dr. Dercum's case in the occipital lobe. Dr. McCarthy decided that this tumor had absolutely nothing to do with the general symptom complex of the case. Therefore nothing was done.

Dr. Tom A. Williams spoke of a case under his care where a large growth on the vertex both bulged outwards and inwards, causing deformity, headache, and mental obtusion. The Roentgen rays showed that the growth extended at least an inch below the inner table. When removal was attempted the hemorrhage was so great that the surgeon had to cease. The portion excised resembled osteoangiosarcoma. Therapeutic application of Roentgen rays has caused subsidence of the headache and mental obtusion, the improvement has lasted six months but treatment is continued.

Dr. S. D. W. Ludlum said he was reminded of a case of Dr. Lloyd's at the Methodist Hospital with a tumor at the seat of the pineal gland. It had caused a great deal of trouble. It was equally well seen in the x-ray picture. It was a psammoma.

Dr. Spiller said in regard to Dr. Lloyd's case it was not surprising that the tumor photographed so beautifully as it was filled with corpora aranacea, and it had been difficult to make sections of it.

NEOPLASM INVOLVING PITUITARY AND SELLA TURCICA TREATED BY "X" RAYS

By Tom A. Williams, M.B., C.M., Edin., Washington, D. C.

A West Virginia woman, aged 37, was referred to Dr. Williams on July 28, because of severe central headaches which had followed a convulsion five months previously. During this period, several convulsions followed by stupor, nausea, extreme dizziness and vomiting occurred now and then; and a diplopia had existed for two months. Her greatest distress, however, was occasioned by the indescribably "awful" feeling, which was constant, and accompanied by a bad taste, she herself saying that this had been present since the death of her mother a year before, and that she had been very nervous and shaky also. She had sharp pains in the calves, felt a weight in the stomach and vague pains all over her at times, and had been losing hair very rapidly. Mercury and potassium iodide had been given for some weeks on account of the history of labial sore six years previous.

Examination showed no diplopia, although she had a blurred feeling in the eyes, a slight haziness of the optic papilla, the arteries of which were very small, and inversion of the visual fields, great exaggeration of the deep reflexes without any diminution of the cutaneous reflexes, great weakness more especially in walking, which was quite unsteady; muscular hypotonia, and subcutaneous hyperesthesia upon deep pressure. The intensity of the headache and the absence of localization symptoms and papilledema, in conjunction with the subcutaneous hyperesthesia and the "awful" feelings, led to the suspicion of pituitary involvement. So the glucose toleration test was undertaken; whereupon it was dis-

covered that from 300 grammes of glucose ingested while fasting no sugar was obtained in the urine. Thereupon the cranium was examined by the Roentgen rays. The plate showed serious erosion of the clinoid processes before and behind, and considerable erosion of the rest of the wall of the sella turcica.

The diagnosis of a growth involving the pituitary body, seemed certain. The treatment adopted was the application of heavily filtered Roentgen rays from a hard tube.

The result was that August 27 the patient felt stronger, walked much better, the blurring of the eyes had diminished, headache was inconsiderable; but the retinal arteries were still over-small, the right disc seemed less clear, and the reflexes continued exaggerated; the visual field had improved. She was sent home for three weeks with the advice that the treatment should then be resumed.

On her return, September 27, she reported that she had felt restless a good deal of the time, had occasional sudden burning sensations throughout the body, and had an almost constant throbbing which she referred to the womb; she had been very nervous and languid, and now and then the eyes felt dim; there had been a beating sensation in the occiput, worst in the morning; her appetite, however, had become good, and the headaches had disappeared.

Examination upon different occasions showed great improvement of muscle tonus, still more exaggerated reflexes, and the blood pressure varying between 130 and 140, with rapid pulse running as high as 138 at times; slight pallor of the left optic papilla without any blur. The visual fields had slightly extended. Her suggestibility was greatly exaggerated so that she was very easily upset by depressing ideas. She was then given pituitary substance, 5 grains twice daily, in the belief that some of the vascular symptoms might be favorably influenced. There is, if anything, an aggravation of the symptoms, and some nausea and further contraction of the visual fields have occurred. Dizziness and pain in the precordia troubled her a good deal, although the pulse was reduced to 90. She is now undergoing the third course of Roentgen ray application.

Dr. Dercum asked Dr. Williams whether his patient had not a preservation of vision and called attention to the fact that Dr. Langdon's patient had no perception of light.

Dr. Charles M. Byrnes read a paper on the intradural administration of mercurialized serum in the treatment of cerebrospinal syphilis.

Dr. F. X. Dercum believed it would be a good plan to try the mercurialized serum and he was glad indeed that a beginning had been made and that Dr. Byrnes had actually achieved results. Dr. Dercum had himself suggested a somewhat similar procedure.

Dr. Ludlum said the same question comes up in relation to intraspinal injection of mercurialized serum as of salvarsan. Why not inject 1/50 of a grain of bichloride directly. Why use the serum? That is a much mooted question but he had never seen it answered satisfactorily.

Dr. Gordon said he had had experience with salvarsanized serum. It is the only treatment he has relied upon lately in nervous syphilis. At the present time in a period of 16 or 17 months his results have been very satisfactory. He cited one or two cases which recently were under his care and which he proposes to put on record. One a musician, a leader of an orchestra in a theater, presented no marked symp-

toms, but just sufficient to make a diagnosis of tabes. He had eye symptoms, loss of knee jerks and of Achilles tendon reflex and some ataxia. Paresthesia in the hands especially annoyed him, so that he could not feel the violin strings that he had to press upon with the finger. He had to give up his occupation for several months and was much distressed. When he came under Dr. Gordon's observation the above treatment was proposed. The musician submitted himself, and it was actually remarkable how the paresthesias gradually and rapidly disappeared and he resumed his occupation. After six weeks a return of knee jerks was observed. In another patient, a locomotive engineer, who suffered sharp, shooting pains in the legs, Dr. Gordon obtained complete removal of the pain in five weeks. The salvarsanized serum he has been using continuously. With one exception the results have been prompt, the paresthesias and subjective disturbances, in particular, have been helped considerably. Dr. Byrnes reports one case in which he said the salvarsanized serum had been used first and this was followed by addition of mercurialized serum. Dr. Gordon believes the salvarsanized serum is an excellent remedy, and it is possible that the addition of mercurialized serum will help the condition. He would like to know how long would Dr. Byrnes have the inunctions or injections of mercury used before he gets the serum. How much mercury he uses before he begins to utilize the mercurialized serum. It is possible that the association of salvarsan and mercurialized serum will give still better results. Salvarsanized serum had been in Dr. Gordon's experience one of the most excellent, if not the best, remedy.

Dr. Williams said he would draw the attention of the Society to the fact that we do obtain excellent results by the treatment of tabes by intravenous injections of salvarsan combined with intravenous and intramuscular injections of mercury. We must not forget the report to the American Neurological Association by Dr. Sachs and Dr. Stearn, who treated by intravenous salvarsan plus mercury, about 137 cases with almost constant benefit.

Dr. Williams has about 60 patients similarly treated, most of whom are functionally well. Many of these are controlled by lymphocyte counts. As the disease was deeply seated in the membranes and the lesions were chiefly around the vessels, access of medicaments must be from the blood and lymph rather than from the spinal cord; besides arsenic is inappreciable in the serum as injected intrathecally by Swift-Ellis technique, while arsenic is found in the ventricles after intravenous injections.

Dr. Charles M. Byrnes in answer to the question why he did not use an inorganic salt, replied that this was best answered by stating what he had abstracted from a paper by Ravaut. The author injected two drops of a 1 per cent. solution of the cyanide of mercury. The result was that the patient had severe muscular spasms of the entire body, and trismus was pronounced. After a few days these symptoms subsided. Dr. Byrnes is of the opinion that the serum preparation is less irritating than the inorganic salts of the heavy metals. In regard to Dr. Gordon's question, he replied that he had had several cases in which the combined serum was used, but did not refer to these cases because of the limited time at his disposal. He is convinced, however, that the mercurialized serum produces a quicker reduction in the cell count in the spinal fluid than salvarsanized serum, and the negative Wassermann

reaction has been obtained in about an equivalent percentage of cases. He does not wish to give the impression that he is not enthusiastic about salvarsanized serum, for he has found it quite effective in many instances, but there are certain cases in which the mercurialized serum is perhaps more valuable.

INVOLUNTARY MOVEMENTS FOLLOWING BILATERAL CEREBRAL LESIONS

By Douglas Davidson, M.D.

The patient, a man 28 years of age, was exhibited for Dr. Spiller, under whose care he was. He had had a rapidly developing left hemiplegia in June, 1914, and after this his mentality was affected and he was childish. The paralysis gradually disappeared. In August, 1914, he had had a second attack in which the right side of the face probably was weak. Following this attack he developed almost purposive movements of all four limbs and involuntary laughter.

When he came under Dr. Spiller's observation he was fairly contented and did not worry about his condition. He answered questions intelligently. When at rest and unobserved, his face was not contracted, but any observation of him, noticed by him, caused a marked spastic smile to appear, which at times became an audible laugh. Speech was slow. The face and limbs on the left side seemed to be a little weaker than on the right side, and the tendon reflexes were somewhat exaggerated. The man was presented chiefly because of the peculiar involuntary movements. When he was at rest these movements were not pronounced, but under observation he performed slow, deliberate, regular to and fro movements of the feet, rubbing the feet along the floor, first pushing one or the other forward and then drawing it back, sometimes giving either foot a sideward motion, or tapping on the floor with the toe or heel. He was unable to control these movements. He had similar movements in the upper limbs; he picked at his coat or trousers, or shrugged his shoulders. He seemed to be under great motor stimulation, as though cerebral inhibition had been greatly diminished, and his movements had a purposive character.

Dr. I. Jones thought the ear situation in this man was a very surprising one. As he understood it the examination of the ear was suggested by the fact that he was deaf; a wax plug was removed from one ear and hearing was completely restored. Examination shows a very excellent degree of hearing on both sides. If it were not for the examination of the vestibular apparatus the ear would be entirely eliminated in this case except negatively. To his astonishment the vestibular apparatus of each side is noticeably impaired and the right is practically dead to the caloric reaction. Definitely speaking, when water 68° was douched into his right ear for four minutes and 25 seconds (until the entire amount of water in the irrigator was exhausted), it failed to produce nystagmus or any deviation in the pointing reactions. If his arm were brought down from above or to the side it would touch every time just as it would spontaneously, if no impulse were carried through. This was done repeatedly on successive days. On the second day after 2 minutes and 25 seconds, a slight result was obtained, whereas a result ought to be produced in 50 or 60 seconds. (A nystagmus should be obtained to the opposite side and a pointing reaction

towards the same side). There was produced at this time the very faintest rotary nystagmus, which could not be observed at all unless he were watched closely and just the least little twitching could be seen. His pointing reactions went the least little bit towards the right, perhaps an inch, nevertheless he distinctly reacted toward that affected side. The left ear showed a more marked nystagmus in 1 minute and 35 seconds, and gave no very marked pointing reactions. All this was observed in a man without any dizziness. It is difficult to reconcile these findings of an obstruction along the line of the vestibular apparatus with a lesion of the lenticular nucleus.

Dr. William G. Spiller said that the case was one of much interest to him. Wilson speaks of an acute onset, in rare instances, of the progressive lenticular degeneration. In Dr. Spiller's case the lesion is not progressive. The disorder is not a family one, but this does not exclude it from Wilson's type. There must be bilateral lesions, one of the lesions may be in the center for the facial muscles and the other near the internal capsule. The symptom-complex is distinctly different from that described by Wilson. The movement is not a tremor. Dr. Spiller had a number of times observed this man when the man did not know any one was watching him, and at such times he was quiet. It seemed as though the movements were caused by some emotional disturbance as a result of removal of inhibition of the brain, so that any excitement caused these unintentional, yet almost voluntary appearing movements.

Dr. Mills remarked that he had studied the patient, not as fully, of course, as Dr. Spiller had, and it seemed to him the case was much as Dr. Davidson and Dr. Spiller presented. He had some little tremor also. He showed it sometimes in his tongue. The movement is similar to that described in some of Wilson's cases. While he has no spasticity, Dr. Mills did not think the tonicity of the muscles was absolutely normal. He had a dystonicity. In fact, the term hypertonicity was not the very best, although so much used in these cases. His speech was somewhat dysarthric, his mental condition was like that in Wilson's disease.

The movements of this patient remind Dr. Tom Williams of the "*forme fruste*" of "*movement de manège*" seen in animals after extirpation of the vestibule; it is as if the patient is about to turn round to one side when the movement is immediately interrupted by the impulse to turn round in the other direction, with the result that he keeps advancing and drawing back one foot and hand after the other, accompanied by a swaying. In conjunction with the other symptoms, it might be that a bilateral lesion implicating the trapezoid body is responsible; for this tract conducts some of the vestibular impulses.

INTENSE JAUNDICE IN THE NEWBORN CHILD AS A CAUSE OF ARREST IN THE DEVELOPMENT OF THE BRAIN

By William G. Spiller, M.D.

Dr. Spiller said he had had four cases of cerebral diplegia with the history of intense jaundice occurring a few days after birth. The ordinary mild icterus of the newborn could not be considered, but in some cases the icterus is severe and it is possible that the cortical nerve cells

might be affected by the intoxication occurring with the jaundice. He had found no mention in literature of severe jaundice as a cause of cerebral diplegia, and he thought it desirable to call attention to this cause. The subject was presented only in abstract form and the paper will be published later.

Case 1.—Child, three years old when seen, was born at full term in normal labor. She was supposed to be a normal child until severe jaundice developed when she was one week old. She probably was unconscious during this attack. The jaundice lasted three days. She was unable to hold up her head until a year old, and unable to rise to a sitting position when lying down until three years old. When examined she presented the appearance of spastic ataxic diplegia.

Case 2.—Child, sixteen months old, was possibly born a little prematurely, in normal labor. Severe jaundice began on the fourth day and lasted about six weeks and the child nearly died. When recovering from the jaundice the head frequently was drawn backward. He had spastic cerebral diplegia when examined.

Case 3.—Child, two years old, was born at full term in normal labor. Severe jaundice developed a few days after birth and for weeks the child was not expected to live. The condition was one of delayed development with hypotonia of the neck muscles.

Case 4.—Child, three years and ten months old, was born at full term in normal labor. Severe jaundice began on the third day and lasted about three months. The condition was one of marked spastic cerebral diplegia.

Dr. McCarthy said he thought this paper of Dr. Spiller's was an important contribution to the subject of cerebral defects and brought to mind some work along these lines that he did some years ago, working out metabolic changes and more particularly the osmotic changes. He did some experiments with rattlesnake venom. A condition of hemolysis developed, associated with an infiltration of iron in the ganglion cells in the cortex.

Dr. Gordon said that if this jaundice was of a hemolytic nature, it is possible that multiple hemorrhages took place in the cortex which subsequently produced softened foci in the cortex, thus causing the arrest. He did not know whether Dr. Spiller had an autopsy or not. That was one of the ideas that came to Dr. Gordon that could explain the encephalic arrest by a pure mechanical process.

Dr. Spiller said in regard to the action of jaundice in the newborn, hemorrhage probably does occur in some of these cases and acts mechanically. He had referred to this in his paper.

COMBINED MEETING OF THE NEW YORK NEUROLOGICAL
SOCIETY AND THE NEUROLOGICAL SECTION OF THE
N. Y. ACADEMY OF MEDICINE

NOVEMBER 10, 1914

DR. SMITH ELY JELLIFFE, President of the N. Y. Neurological Society,
and DR. I. STRAUSS, Chairman of the Neurological Section of the
N. Y. Academy of Medicine, presiding

SCLERODERMA, WITH CONTRACTIONS

By S. P. Goodhart, M.D.

The patient was an unmarried girl, 24 years old, a Hungarian Jewess, whose family and previous personal history showed nothing of interest. Her present illness began about nine years ago, when she noticed a swelling of the toes, followed by increasing stiffness of the toes and knees. About the same time she also complained of a feeling of coldness and weakness in the lower extremities, with paroxysmal attacks of whiteness of the skin of the hands, together with pain and tingling. With the disappearance of the blanching, the hands would become congested and then cyanotic. Because of the increasing weakness the hands lost in function, and flexure contractures soon appeared. These symptoms for a time increased in severity and then remained stationary.

About five and a half years ago the patient noticed pigmentation of the chest and a peculiar stiffness of the face, so that she was scarcely able to open her mouth. Some months later there was some improvement in the power of moving the knees and toes. When the weather was cold she had noticed a numbness of the nose and tongue, but not of the lips nor ears.

The legs could be separated for a distance of only about six inches at the knees, due apparently to fixation at the hip joints. Over the feet there was a hidebound condition, but less marked than in the hands, which presented the typical appearance of a pronounced sclerodactyle. The face was distinctly masked in character, with all lines obliterated: the palpebral fissures were wide, giving a strange expression to the eyes. The margins of the lips were very narrow, and the mouth was held stiffly and always partially open, giving rise to a mummified expression. The nose was narrow and angular. The ears, which were indurated, thin and blanched, were held rigidly down. The mucous membrane of the mouth was becoming involved, showing some induration to the touch. The scalp showed no abnormality. The mammae were fairly well developed. Trophic disturbances were well marked over the small joints of the fingers and toes. The skin showed glossiness as well as the pigmentation. Many scars were present, due to previous ulcers of the fingers and toes.

Sensation.—A few years ago there was a slight but manifest diminution to painful stimuli from the finger-tips up to the wrists: this was also true of the feet and ankles. At the time of examination no sensory changes were found excepting that there was a lack of proper appreciation of hot and cold stimuli.

Dr. Goodhart said the interesting features in this case were the initial symptoms, which pointed strongly to Raynaud's disease: indeed, that was the original diagnosis and the one with which the patient was discharged

from the Harlem Hospital. Further, the case offered a recognition of the various stages of the disease, so far as it affected the soft tissues, namely: edema, induration and atrophy, and the characteristic cutaneous pigmentation. The restriction in the movements of the joints was by no means entirely due to the hidebound condition: there were true changes in the joints giving rise to ankylosis. The hip joints were so restricted by the intra-articular pathological process as to afford very little movement to these joints. There was doubtless rarefaction and absorption of osseous tissue in some of the larger joints analogous to the atrophic process of the subcutaneous tissue.

There had been periods of marked improvement in the condition of this patient, and for a time the administration of thyroid extract seemed to be responsible for it. The initial symptoms affecting the periphery of the extremities and suggesting Raynaud's disease, together with the dissociated sensory changes, indicated a trophoneurosis. An interesting symptom in this case was the beginning involvement of the mucous membrane of the mouth. The vaginal mucosa had not been examined.

Dr. Goodhart said he did not believe that the pigmentation in this case was really indicative of adrenal involvement, there being no other symptom of Addison's symptom-complex.

Dr. Walter Timme thought this condition was one of generalized connective tissue hyperplasia, of which the scleroderma was only a single manifestation. This connective tissue hyperplasia, in turn, was secondary, as a rule, to a disturbance of the sympathetic and autonomic nerve supply. In most of these cases of scleroderma we get the history of an antecedent high grade sympathicotonia, and as this increases we begin to get the tissue hyperplasia, sometimes in the muscles and joints, and occasionally in the thyroid, the hypophysis cerebri and blood vessels. We have an overactive sympathetic and an underactive autonomic system. In the end, it probably resolves itself into a question of disturbance of the internal secretions, and from a therapeutic standpoint we had seen some benefit from the use of various internal secretions. These must be brought to bear upon the particular symptom that was in the foreground, and it required a good deal of application and patience to arrive at any result. Many of the symptoms of these patients, such as the lack of perspiration, the occasional rapid pulse, etc., went to prove the contention that the condition was due to a disturbance of balance between the sympathetic and autonomic nervous systems.

In reply to a question as to whether he regarded the lesion in the case shown by Dr. Goodhart as cortical or subcortical, Dr. Timme said it had been shown that the ablation of one of the hemispheres gave rise to certain sympathetic manifestations, but just what part of the hemisphere was involved we did not know. The highest level to which they had been traced thus far was in the region subjacent to the corpora quadrigemina, to the hypothalamus, to the stalk of the hypophysis and some of the centers of the vagus. Manifestations of the autonomic system had been found in hypophysis stimulation.

Dr. Goodhart, replying to Dr. Timme, said that in spite of the lack of pathological evidence, the clinical evidence seemed to be very striking of a higher center, probably about the motor area.

A CASE OF DYSTONIA MUSCULORUM PROGRESSIVA

By H. Climenko, M.D.

A girl, eleven years old, born in Russia of Jewish parentage, was admitted to the Montefiore Home on September 27, 1914, with the following history: Her father, who was 43 years old, was not addicted to the use of alcohol and smoked moderately. Fifteen years ago he had an attack of acute articular rheumatism. He denied any venereal infection. The mother was healthy. She had had one miscarriage and four children at full term, who were living and in good health.

The parent, who was the third child, was born at full term after a perfectly normal labor. She was breast fed and began to walk and talk at about the age of eighteen months. The first tooth was erupted at five months. She had German measles when she was two years old and a year later had two attacks of tonsillitis. When she was nine and a half years' of age she had an attack of whooping cough. The bowels had always been irregular; her appetite was poor and she was frequently restless in her sleep. She played a good deal out of doors and was considered bright at school.

About a week or two after the attack of whooping-cough, *i. e.*, about eighteen months prior to her entrance to the Montefiore Home, she began to complain of weakness in her left lower leg. A little later she noticed that the foot turned outward while running or walking quickly. The weakness gradually extended upward, and her knee gave way. Plaster-of-Paris was applied to the knee joint, which seemed to have relieved her a good deal. A few weeks later she began to complain of weakness in the opposite leg, and soon the left hand became involved, and with each attempt to use the hand it began to tremble. Similar tremors were also noticed in the lower extremities. She complained of no pain nor paresthesias of any kind. The condition progressed steadily, and about a year after the onset of her symptoms she was no longer able to walk.

Her chief complaints at present were inability to walk, weakness in the right upper extremity and trembling of all extremities. In the recumbent posture the patient showed a slight wormlike tremor of all the extremities, more pronounced on the right side. In a position of rest, with the extremities as nearly in repose as possible, this tremor, though constantly present, was better appreciated by touch than by sight. It somewhat resembled an athetosis, but lacked all the characteristics of that movement. It seemed to possess a characteristic of its own, being somewhat irregular and wormlike, and entirely lacking in rhythm. Perhaps the word dyskinesia most appropriately described it. In the prone position there seemed to be a characteristic pose consisting of flexion of the right knee, with extension and external rotation of the right hip, and outward rotation of the left foot. There was a slight kyphosis in the lumbar region which became more pronounced in the sitting posture. On standing, a kypho-scoliosis was equally pronounced. The trunk assumed an acute angle, with the pelvis of the latter varying in its position with movement. The patient could stand only with support. On standing, the right foot was at an unusual angle with the lower leg, the right knee assuming almost the shape of a genu recurvatum. The patient was unable to walk unsupported, a marked feature being a peculiar torsion of the trunk on the pelvis, the latter being thrown out of a normal angle rela-

tion with the trunk. On trying to walk there were numerous unskilful and disharmonious movements through extremities and trunk: to correct these movements, the patient seized her lower extremity at the joint, forcing it into proper position. She thus expended an inordinate amount of energy in attempting with her hands to facilitate locomotion, alternately pressing back one knee and then the other as the obstreperous joint gave way under the body weight.

The right hamstring muscles were in a state of hypertonicity; almost rigidity. The muscles of the right leg were in a similar condition. Both quadriceps groups and the left peroneal group were, on the contrary, in a state of complete atonia. The hips were hyperflexed on the abdomen. Due to the atony of the flexors digitorum of the right hand, the fingers could be hyperflexed to such a degree that on dorsal flexion (really extension) of the fingers, they could be brought to an acute angle with the dorsum of the hand.

Both knee jerks were diminished, and the ankle jerks were obtained with difficulty. The Babinski and Oppenheim signs were absent. The abdominal reflexes were present. All the various sensibilities were intact. The cranial nerves were normal. The heart, lungs and abdominal viscera showed no abnormality. The patient was able to write with difficulty. The electrical reactions showed no qualitative changes: a hyperexcitability, however, was obtained in the hypertonic muscles. The urine showed a slight excess of indican, but was otherwise negative. The psyche was perfect. The x-ray showed that the neck of the left femur was shorter and thicker than the right and the sacrum was turned to the left. There was no change in the density of the bone.

Dr. C. C. Beling, of Newark, N. J., said that at a meeting of this Society some time ago he presented a case bearing some resemblance to the one shown by Dr. Climenko. That was the only case he had seen. The one shown tonight seemed to be in a rather earlier stage.

Dr. I. Abrahamson said he showed a case of dystonia musculorum progressiva at a meeting of this Society about two years ago, and since then he had seen two additional cases. In the case he had presented, the disease had previously developed in the patient's sister, and these were the only instances on record, so far as he knew, of two cases in the same family.

Oppenheim, in his description of this disease, spoke of its usually beginning in the hand, the child noticing that it could only write with difficulty, the symptoms later extending to the leg, etc. In a typical case which came under his observation about a week ago at the Montefiore Home, the patient, a girl, had hypertrophy of the lower extremity which was affected by the dystonia, and from time to time there were cramp-like spasms in these muscles. This hypertrophy and hypertonicity is also observed in Thomsen's disease.

The case shown by Dr. Climenko tonight, Dr. Abrahamson said, seemed to present a typical case of dystonia musculorum progressiva.

Dr. I. Strauss said that in one case that came under his observation, in addition to the hypertonicity, there was reaction of degeneration in the muscles of one leg. That patient died. No autopsy was obtained. The condition began in one arm following a trauma, and the first appearance of the tremor was in the injured arm. From here it spread over the entire body, and the child was in a constant state of spasm, so that repeated doses of hyoscine were required to keep it quiet. In this case the dystonia and lordosis were extreme.

Dr. William M. Leszynsky said that the case of dystonia musculorum progressiva which he showed at a meeting of the N. Y. Neurological Society about nine years ago was the first case of the kind presented in this country. That patient had been subjected to various forms of treatment, including tendon cutting to relax the muscular spasm, etc. At a later date he was again shown at a meeting of the Society by Drs. Hunt and Krauna. Dr. Leszynsky said that at the time he presented the patient, no diagnosis was made. There was no scoliosis, but the boy moved about on all-fours, with constant peculiar and fantastic movements. He improved for a time, but subsequently his symptoms became exaggerated.

Dr. Climenko, in closing, thought that Oppenheim emphasized the fact that there were no organic muscular changes, hypertrophy or atrophy, and that the characteristic marks of the disorder were hypertonia and atonia: these he regarded as the only cases that should come under the category of dystonia. Other writers on this subject failed to agree with Oppenheim, and cited cases in which atony was lacking.

PILOUS CEREBRAL ADIPOSITY

By Walter Max Kraus, M.D.

Many instances of genital dystrophy associated with obesity have been described since Froelich first separated the syndrome from the general class of obesities, but Dr. Kraus had not found any account of a class of cases complicated by an anomalous condition such as was presented in this case; namely, a marked increase of the body hair, accompanied by a lack of the dry skin characteristic of the classical Froelich syndrome.

This patient, in the service of Dr. Alexander Lambert, in the medical wards of Bellevue Hospital, was a white man, 31 years old; a watchman by occupation. His father was living and an alcoholic: his mother died of nephritis following childbirth. One brother died in infancy of unknown cause: one sister was living and well. Otherwise, the family history was negative. The patient had been married since 1906. His wife had had one miscarriage and four normally born children: three of these had died in infancy, one of meningitis, the others of unknown causes. The living child was two years old and well.

The patient had been accustomed to drink from five to seven glasses of beer daily up to the onset of his present illness in 1910. He smoked moderately. He gave a history of measles, chicken-pox, diphtheria and whooping-cough in childhood, and some form of arthritis from the age of 24 (1908) until one year ago. The soles of his feet were sore and later the tissues about the knees became red, swollen, hot and painful. After several of these attacks there was some limitation of motion, but one year ago, after treatment at the Metropolitan Hospital in this city, the patient was cured.

Between the ages of three and four years he fell from the fifth story of his residence to the ground, resulting, as he states, in a fracture of the crown of the skull. He was unconscious for about 14 hours and disabled for six months. From that time until the age of thirteen he was entirely well. Then he began to have attacks of Jacksonian epilepsy, which occurred from three to six times yearly and were not of sufficient severity to cause the patient to either tell his parents or seek the aid of a physician. At the age of 25, a little over twelve years after the first attack, the attacks ceased and had not since recurred.

Four years ago, at the age of 27, the patient began to grow fatter. At that time his weight was 150 pounds; in 1912 it had increased to 204 pounds; in 1913 to 263 pounds, and his weight at the present time was 282 pounds. At about the same time as this increase in adipose tissue began, the following symptoms made their appearance: his hair began to increase over the trunk and extremities, he felt sleepy and as time went on he would fall asleep involuntarily in addition to sleeping a good deal voluntarily. In 1910, while driving a large automobile beer-truck, he fell asleep, allowing the truck to run into a saloon. This accident deprived him of his chauffeur's license. These narcoleptic attacks had continued since. The patient's appetite for food, especially meat and carbohydrates, had increased. He also complained of shortness of breath and sweated a good deal. Three and a half years ago he noticed that his penis was smaller and that shortly afterwards his sexual desire grew less until now he had none at all.

Dr. Kraus said it was difficult to form any clear conception of the basic cause in this case. A fall on the head at four years of age, followed nine years later by mild Jacksonian epileptic attacks which lasted thirteen years led one to believe that some neoplasm was present. The beginning increase in the accumulation of fat two years later pointed to an involvement of the glandular hypophysis, with the probability that in the interval the glandular part was gradually decreasing in power until its limit of safety was overstepped, with the ensuing appearance of the Froelich syndrome, complicated by atypical changes in the skin and blood. The absence of any involvement of the cranial nerves or of a cranial neoplasm made it impossible to settle on any diagnosis other than hypopituitarism.

A blood examination showed 6,720,000 red cells; 10,800 white cells; polymorphonuclear neutrophiles, 56 per cent.; eosinophiles, 3 per cent.; transitionals, 1 per cent.; large lymphocytes, 19 per cent.; small lymphocytes, 11 per cent.; -large mononuclears, 10 per cent.; hemoglobin, 110 per cent. (Sahli).

Urine: Albumin ranged from one to six grams per day during two weeks observation; usually about three grams. No sugar. Many granular casts and a few white blood cells.

Carbohydrate tolerance: From 100 to 500 grams of dextrose were all without effect in causing glycosuria. Seven hundred grams were followed by only a trace of sugar in the urine.

Blood pressure: Systolic, 170 mm.; diastolic, 120 mm. Wassermann negative.

Dr. Abrahamson said that in the wards of Mt. Sinai Hospital they had at the present time a young woman with very similar manifestations to those in the case shown by Dr. Kraus. She gave a history of amenorrhea, hyperidrosis, peculiar skin changes, adiposity, she complained of sleepiness, had a growth of hair on the face and body, but the urine contained sugar and showed as in Dr. Kraus's case evidences of an advanced nephritis. A blood count gave 6,100,000 red cells and 10,000 white cells, with 110 per cent. of hemoglobin. The facies looked like a typical one of polycythemia. It was noteworthy that the urine constantly contained a small percentage of sugar. The sugar tolerance was being investigated. The moist skin in this case might be attributed to the nephritis, as well as the polycythemia, but was most likely a part of the polyglandular disease. As he had not seen the x-ray plate, he could not report on the findings.

Dr. Strauss said that in the case mentioned by Dr. Abrahamson the x-ray showed an enlargement of the sella turcica which was enough to be due to a tumor.

Dr. Timme said that the hypertrichosis present in this case was not quite as generally distributed as had been stated, for upon closer inspection it could be seen that the distribution was radicular in character, and furthermore skipped the areas supplied by the cervical roots to begin again at the dorsal level. This arrangement corresponds with the position of the cells of the superior spinal sympathetic nucleus, which is non-existent in the cervical region of the cord, and begins at about the first dorsal level. This is rather suggestive, therefore, of sympathetic involvement.

Translations

VAGOTONIA

A CLINICAL STUDY

By PRIVATDOZENT DR. HANS EPPINGER AND DR. LEO HESS

OF VIENNA

TRANSLATED BY WALTER MAX KRAUS, A.B., M.D., AND
SMITH ELY JELLIFFE, M.D., PH.D.

(Continued from vol. 42, page 119)

Several observations on conditions not typically Basedow's disease deserve mention. These conditions are of thyroid origin, but are not fully-developed classical thyreopathies. They have been designated as "goitre heart," or as "Basedowoid Conditions" (Stern²⁸).

Since the symptoms of vagotonia (diarrhea, subjective palpitation, sweating, eye symptoms, tremor and general nervousness) are very similar to those of thyroid intoxication, the possibility must be advanced that certain questionable and atypical forms of thyroid disease may in reality be vagotonia. It is a frequent occurrence to diagnose Basedow's disease on the basis of one cardinal symptom,—as tachycardia—without determining the presence or absence of the main characteristic symptom,—increased metabolic exchange, with resultant loss of weight.

If this be made the crux of a diagnosis of Basedow's disease, it will be found that many cases of so-called incompletely developed Basedow's disease are in reality nothing but vagotonic neuroses. If these cases are operated upon it will be found that, as a rule, no good effect will be obtained, indeed there may be some aggravation of the disease. As to the symptomatic picture of cases of "goitre heart," we cannot fail to observe that the vagotonic picture is so conspicuous that there seems to be but a combination of vagotonia and goiter. The coëxistence of two

²⁸ Stern, Differenzialdiagnose und Verlauf des Morbus Basedowii, Jahrbuch d. Psych. u. Neur., 1909, Vol. 29.

pathological, constitutional states, vagotonia and endemic goiter, must be thought of particularly in neighborhoods where goiter is endemic and where Basedow's and hyperthyroidism are known to be relatively infrequent (as in Styria and certain Alpine districts).

Some observations must be made upon the changes at the climacterium. This state gives rise to many nervous symptoms; some of cardiac or vasomotor origin, some of gastro-intestinal and metabolic origin. Analogous to these are the withdrawal symptoms after castration, when vasomotor disturbances are also to be found. Not infrequently one finds that there is an enlargement of the thyroid at the climacterium or during pregnancy. It is a simple deduction to suppose that when vasomotor or visceral symptoms coexist with thyroid enlargement, a similar disturbance exists in the relations of the endocrinous glands, as undoubtedly occurs in Basedow's disease. If one analyzes the symptoms of the climacterium, one finds it easy to relate them to the symptoms produced by stimulation of the autonomic nervous system. The symptoms include unaccountable sweating, v. Gräfe's sign, marked dermatographismus, hyperacidity, and hypersecretion, colic-like pains about the pyloric or gall tract region, and high sugar tolerance. Other symptoms, most frequently observed in vagotonia, are respiratory disturbances and cardiac pains, associated with symptoms like those of angina pectoris vasomotoria.

The metabolic change, the post-climacterial obesity, may be contrasted with the emaciation resultant upon sympathetic stimulation (adrenalin poisoning). As a general rule vagotonic stimuli are most often observed in youth. It is therefore a matter of surprise to find disturbances as gastrosucchoresis and pylorospasm in elderly men between forty-five and fifty-five. It would seem that men, like women, show symptoms of a climacterium, symptoms due to an absence of the sex-gland secretion, with consequent furtherance of autonomic stimulation.

Juvenile emphysema deserves a word of mention in connection with the relation of vagotonia to disease symptoms. Quite justly this disease is attributed to some disturbance of the respiratory mechanism. When the lung is distended for a considerable period by increase in the rate or the degree of inspiration, which almost exclusively serves the process of expiration, the passive contractile elastic apparatus is very materially overworked, and may eventually become insufficient. An increased tone of the

bronchial musculature will make the expulsion of air by expiration difficult and will cause a lasting distention of the lung.

Analogous to this is the diminished power of the elastic elements of the lung to expel the alveolar air by expiration, a condition which develops upon a chronic asthma, or a chronic bronchitis, and is due to relative narrowness of the bronchial tree.

Investigations carried out with the spirometer and described in a previous section have shown that in these cases we have to do with changes in the tone of the bronchial musculature. We maintain that increased tone in these muscles possibly aggravated by a coëxistent bronchitis, will, when combined with an insufficiency in the respiratory mechanism, pave the way for juvenile emphysema.

In confirmation of this theory, we would say that we have never failed to find signs of vagotonia in cases of juvenile emphysema. We have no clinical observations upon thymic asthma or laryngospasm of the newborn. It seems, however, that the mechanical element, compression by the thymus is not the only factor in their production, but that nervous disturbances, perhaps related to the thymus secretion, play a rôle in their etiology. We would suggest atropin for their relief.

It is no less interesting to give some attention to the nervous element of certain skin diseases, particularly those involving the blood-vessels. We, ourselves, have only studied urticaria, and as far as this group is concerned we can state that vagotonic manifestations are very conspicuous. We found high grades of hyperacidity, greatly increased gastric tone, almost a pathological state of peristalsis, and in most instances, eosinophilia.

Sweating is frequently complained of and hyperidrosis and troublesome salivation often result from the administration of pilocarpin. These individuals do not react well to adrenalin, and have a high sugar tolerance.²⁹

Bronchial asthma and urticaria are not infrequently associated. The gastric changes did not give rise to symptoms in our case, and were in the background. When we consider that the vasodilators are controlled by the autonomic nervous system, and that the dermatographism seen in vagotonics may develop into an urticaria, the parallelism between vagotonia and urticaria becomes very striking.

²⁹ *Translator's Note.*—This statement has not been borne out in our experience, since 1 c.c. of adrenalin has been found to dispel some urticarial rashes in an hour or less.

A few observations upon anaphylaxis seem pertinent to our subject. If one inject a small amount of horse serum, and repeat the injection after three weeks have elapsed, severe anaphylactic phenomena result: a general eruption, collapse, dizziness and vomiting. The newer researches on the subject have shown that the symptomatic phenomena of anaphylaxis are due to autonomic irritation, and that these phenomena may, in some animals, at least, be relieved by atropin. These facts make it desirable, both for theoretical and practical reasons, to study the manifestations of anaphylaxis in vagotonic individuals particularly since we know that serum disease does not occur in all cases in which it might be expected. Isolated observations from various sources have been of great interest to us. For example, the injection of serum and particularly its re-injection in children who have been asthmatic or have had laryngeal spasm (spasmodic croup) has been found to be very dangerous. Since we feared acute anaphylaxis in our vagotonic patients, and wished to avoid the general symptoms, which it produces, we gave our second injections subcutaneously, and only observed the occurrence of local changes. Of thirty cases taken at random, only three showed any signs of a reaction. These three were vagotonics. Immediately after the injection, a large papular elevation formed about the puncture in the skin. This grew steadily larger. In the course of the next twenty-four hours, a general and very irritating urticaria developed.

In those who were not vagotonic the greatest reaction seen was the local one and this was absent in many of the cases. Further study of this question seems to us to be very desirable, not only on account of the dangers of serum disease, but also on account of its prevention by atropin. This study would seem to promise most if applied to children.

That the symptom, fever, lies in close relation to sympathetic stimuli is readily seen. Adrenalin will cause a rise in temperature, and cocain, which increases the action of adrenalin, may, according to the researches of A. Fröhlich, cause fever.

There is also much evidence to show that the various vagotropic substances have a temperature-reducing action. We also know that certain of the antipyretics cause some vagatonic symptoms as sweating and lowered blood pressure.⁸⁰

⁸⁰ *Translator's Note.*—See editorial, Jour. Amer. Med. Assn., Vol. 62, p. 302.

It may be assumed that the relationship between vagotonia and fever is such that vagotonics will tend not to develop as high fever as those who are not vagotonics. It is well known that there are individual variations in the fever reaction in that some will develop a high fever after minor and insignificant infections. We may say that our study of vagotonics has not shown any parallelism between vagotonia and lessened tendency to develop fever. The only thing worth noting is a fact which has been mentioned before, *i. e.*, that those with sympathetic Basedow's have a greater tendency to rises in temperature than those of the vagotonic type. They are the group which react with high temperatures to mild infections. An attempt to prove a relationship between vagotonia and infection should certainly be made in the near future.

The clearest relations are those of tuberculosis. At the onset, many cases of tuberculosis show various signs of vagotonia, as a tendency to sweat, diarrhea, lowered blood pressure, stomach and intestinal disturbances. Furthermore the pharmacological tests with adrenalin and pilocarpin showed that scarcely any case of tuberculosis reacts to the former, while they always react with typical symptoms to the latter. A group of cases, which showed no signs of Addison's disease, did, however, show an increased sugar tolerance. This raises the question as to whether the toxin of tuberculosis does not have a specific predilection for the chromaffin system. Corresponding with this idea are certain findings in the adrenals in severe forms of tuberculosis. Though these glands show neither microscopical nor macroscopical changes, functional tests show that they contain very little or no adrenalin. There is also evidence to prove that scrofula, which is related both to tuberculosis and the lymphatic constitution, is associated with an over-irritable autonomic system.

Later we shall deal more in detail with Addison's disease and its relation to the lymphatic constitution, since in it the vagotonic signs and symptoms are most clear cut and are undoubtedly due to an absence of the adrenalin producing organs.

At all events, our few observations upon infectious diseases will make it easier to establish upon an experimental basis the etiological relationship between the vegetative nervous system and the disposition to infection.

(To be continued)

Periscope

American Journal of Insanity

(Vol. LXIX, No. 5 (Special Number))

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This number is devoted to addresses delivered at the opening of the Phipps Psychiatric Clinic, Johns Hopkins Hospital, Baltimore, Md. April 16-18, 1913.

2. *Address of the President of the Board of Trustees.*—Of general character.

3. *The Psychiatric Clinic and the Community.*—Review of the general aims of the clinic and its relation to the community.

4. *Specialism in the General Hospital.*—Historical and reminiscent, with suggestions as to the place of Psychiatry among the specialties in the medicine of the present and future.

5. *The Psychiatric Clinic.*—A sketch of what has been accomplished and what problems stand before the Psychiatric Clinic in the immediate future, especially emphasizing its function of investigating the beginnings of morbid mental habits and beginning psychic disturbances and of initiating methods of prophylaxis of mental disorders.

6. *Sources and Direction of Psycho-physical Energy.*—According to the purely physiological view the functions of the nervous system are made up of those of its individual neurones, each an irritable conductor which

reacts to any stimulation applied to it, and passes on a wave of excitement to all its parts. When it is in physiological connection with another cell or cells this wave of physico-chemical excitement acts upon these cells and provokes in them a similar reaction. These functional units are then regarded as "linked together on the plan of reflex arcs of various degrees of complexity through which physico-chemical change propagates itself according to the relative irritability or openness of the various paths thus constituted." According to this view the mental accompaniment of cerebral process, consists in the successive and simultaneous appearance in consciousness of sensory elements or sensations of many different qualities each being as it were attached to one of the higher or cerebral reflexes. Attempting to apply this conception to the cerebral processes involved in the higher modes of mental activity, the author soon found it inadequate and has found it necessary to supplement it with one which he calls the "principle of the vicarious usage of nervous energy." According to this conception, the energy converted from potential to active within each neurone upon its stimulation, must not be regarded as confined to this neurone, but capable of flowing on into and through other neurones from place to place in the central nervous system. This energy may well be denominated "neurokyme" (Forel) or psycho-physical energy. The whole afferent side of the nervous system especially the cerebellum and the basal ganglia of the cerebrum must be regarded as a great common reservoir of free nervous energy (neurokyme) from which all the various efferent channels can draw in turn. He says: "within the many parts of this common reservoir the quantity or potential of free energy must be conceived as varying from moment to moment according to the degree of stimulation to which it is exposed and according to the freedom of escape permitted to it, by the ever varying conditions of tension within the whole system." "But owing to the abundance of interconnection, the freed energy resident in each part can under favorable conditions be led in part at least to any of the many efferent channels, there to be vicariously used for the production of reinforcement of executive activities. Such vicarious usage, it seemed to me, is involved in all processes of concentrated effort, whether of bodily movement or of attentive perception or thought." This notion of vicarious usage of freed nervous energy is also supplemented by the subsidiary hypothesis of the "common reservoir" of freed energy and that of "inhibition by drainage." The acceptance of this group of notions seems to go far towards explaining the fact that the nervous system works as an organic whole. This all however provides a mechanism only and for completion of the scheme it is necessary to recognize a peculiar function of organism "best expressed by the word conation" or will in the "broadest sense of the word, effort towards or striving after ends."

Observation of the behavior of animals shows that the activity of each species is directed mainly toward a small number of special ends—reproduction, securing food, escape from danger, protection of the young, with the violent destruction of whatever opposes these great tendencies. The concentration of an animal upon one of these ends does not depend upon acquired experience, but upon an innate tendency which we call an instinct. Consideration of human behavior shows also that man is endowed with similar tendencies by means of which the great ends of his activities are chiefly determined. "Whenever an organism, whether man or animal is placed in a situation which appeals to one or other of its instincts, which

excites or brings into activity one of these great conative tendencies common to the species, that organism acts with vigor and persistency; all its movements and attitudes imply a great liberation and expenditure of nervous energy, which energy is concentrated towards securing the instinctive end, and yet in its superabundance tends to overflow its prescribed channels and to suffuse the whole organism. And what we call emotion appears to be the bodily and mental expression of this great liberation of nervous energy, concentrated primarily in certain nerve channels specific to each instinct, but secondarily overflowing and diffusing itself through the whole or a large part of the system." Each instinct seems then to be in a sense a "spring of psycho-physical energy."

After considering the view that the neural nucleus of each instinct is made up of a group of neurones which are capable in a much higher degree than the others of storing up energy and discharging it, the author has found it unsatisfactory and has found the idea that "the great outburst of nervous energy which occurs upon the excitement of an instinct to activity, represents not so much the energy actually liberated by the katabolism of specialized neurones forming the kernel of the instinct, but rather energy liberated over a wider area and in some manner gathered together, concentrated, and directed to specific efferent channels, by the instinctive disposition." "May it not be then, that the essential kernel of each instinct seated in the basal ganglia is a wide channel (or one of high conductive capacity) upon which many channels converge both from below and from above (from the cortex of the great brain)?"

Considering another feature of the subject, experimental researches, especially those of the Wurzburg schools have made clear the influence of the intention of the subject, that is the task to which is set the determining tendency, the attitude of consciousness that is the "conation." For instance, if a list of say twelve nonsense syllables is read in an attitude of indifference, 150 repetitions may fail to form sufficiently strong associations to enable us to freely reproduce the series, while if the will is bent to the task it may be committed to memory in as few as 10 or 12 readings.

A mental process is effective in establishing associations in proportion to its involving strong desire or volition, which on the neural side means effective concentration of psycho-physical energy. The experiments mentioned not only justify the notion that conation involves special liberation of energy, but they also compel us to recognize that conscious conative effort once made, the conative process may continue to work subconsciously for an indefinite period of time. In this way laboratory researches tend to bridge over the gap between the older normal psychology and the newer psychopathology, the most important feature of which is the great influence attributed to subconscious processes. While the author is not prepared to subscribe to all the tenets of the Freudian school he holds with Bleuler, that its fundamentals are continuous with and are developments of psychological truths ascertained by other methods than psychoanalysis. The affectively toned "complex," whether repressed or not, has a functional reciprocity with one or more great conative dispositions. The Freudian notion of the transference of affect and that of the sublimation of a tendency he finds also abundantly demonstrated by the observation of normal mental processes, without resort to psychoanalysis, and fitting in with the scheme of neural functions which he has outlined. Freud's view of the development of the sexual instinct also harmonizes well with this scheme,

though the author cannot agree with the view that every other human instinct is but an offshoot from it.

7. *Autistic Thinking*.—Beginning with the story of a schizophrenic who while he is ordinarily well behaved, works during the week and enjoys himself on Sundays like the average quiet asylum patient, suddenly getting the idea that he is to marry the Queen of Holland enters a country inn where he is to meet his bride and can only be removed by force, the author develops the fact that the thinking of this unfortunate, who has never known aught but misery, is not different in its inception from the fairy tales with which children and even adults have always indulged themselves, only this man does not read a fairy tale or tell one but attempts to live one. Each of us has his fairy tale and while under ordinary circumstances we only indulge ourselves in secret and conceal our phantasies from the public view, they nevertheless influence our actions far more than we are willing to admit or even realize, so that the difference in manner of thinking between the normal man and the lunatic is mainly a relative one, and with us all, thought is often divorced both from logic and reality. This form of thinking has been called by the author "autistic" "corresponding to the idea of schizophrenic autism, which turning away from reality sees life in fantastic pictures, and is founded precisely upon autistic thinking."

Psychology has only recently turned from the study of logical thinking to that of the autistic form, the knowledge of whose laws "alone throw light upon the important but hitherto almost ignored inner life of the individual, upon the influences which shape the creations of the poets, and upon those which impel the peoples in their formation of their views of custom and religion, and in their dealing with home and foreign politics." The child ignores reality to a very high degree, he "plays" that so and so is the case and for him person, animal or object assumes successively many rôles. The true poet is in many ways like the child and upon his ability to develop his own phantasy and to transfer his own feelings to the characters whom he creates, rests his greatness and truth to nature. "Poetry is closely related to mythology and between the two stands legendary lore, the formation of which is naturally a very complicated one." We know that behind many of the heroes there are no real persons for the German Siegfried and the Jewish Samson are really sun gods. "The greater part of mythology is really symbolism." "The times of the year, the fructifying and scorching power of the sun, the animal procreative power and similar things are represented as human characters and actions." "The process even more than in the poet is an unconscious one. The myths are understood literally." All this is in direct contrast with the logical point of view according to which all these notions though beautiful and poetic are nonsense. "Our human understanding can never compass everything; we can therefore draw no hard and fast line between the two forms of thinking but the more knowledge we possess the greater will be the possibility of logical thinking." "As a rule the symbolism of the normal individual replaces the conceptions which are difficult to represent and to conceive of, or against which there is a resistance. Especially we like to replace abstract by concrete ideas." For instance a whole railway system will be represented by a wheel with wings, a serpent will represent sin." "In dreams and morbid conditions symbols will not only be used more frequently, but they will be extended to embrace even easily representable things. A patient maintains that she is Switzerland and the Cranes of

Ibycus, which appears at first sight as nonsense. Upon more careful consideration however a certain sense is found in these assertions. The patient is confined against her will in an asylum, she desires to be free. Switzerland is a republic, a free country, she wants to be free, to be like Switzerland and finally she is Switzerland, Helvetia (the figure on the Swiss coins). She is moreover the cranes of Ibycus, that is, she has (in the lines of Schiller's poem)

"Kept free from guilt and failings
And pure her childlike soul."

"A particular form of autistic idea is the systematic shifting of sexual conceptions to other parts of the body, more especially upwards." "In the fairy tale as in the crazy ideas of the schizophrenic many a child is born from the mouth." Delusions of poisoning in women sometimes originate from their desire for offspring. "They believe, as in fairy tales, that they will become pregnant from the food they are given, and some supposed admixture with the food is symbolized as poison." "In autistic thinking the grossest contradictions may exist side by side" as well within the thought itself, as in its contradiction of the representations with the outside world. As the child may be at the same time "little Jack" and a great general so the insane man may be Brown and at the same time Emperor, Pope or God Almighty. As long as the insane are composed in their thinking they are partly conscious of the contradictions with what sound people call reality. So long as they are in need of causal connections they must seek an explanation for such contradictions. The man who has a delusion of great wealth may realize that he is not enjoying it, hence develops the idea of having enemies who are keeping him from his possessions, are persecuting him. "Autistic thinking has special connections with sexuality." And schizophrenic patients may enjoy the delights of love through hallucinations, though here the feeling of satisfaction is apt to be mixed with something unpleasant, the feeling of sinning against nature. Many desires are connected at the same time with agreeable and with disagreeable feelings, that is they are ambivalent. This ambivalency leads even in normal people to difficulties of decision and inner conflicts. Still more is this the case with schizophrenics in whom ambivalent tendencies seem frequently at the root of their delusions. A certain degree of ambivalency is always connected with sexuality and psychoanalysis shows that almost all men have at least in a rudimentary degree abnormal sexual tendencies, though the healthy person is scarcely conscious of them and overcomes them without effort.

This is noticed in the Oedipus-complex, and it was known even by the Greeks that this story was the expression of a universal tendency, as psychoanalysis shows to-day that there is a certain admixture of sexuality in the love of children for their parents of the opposite sex. "Autistic thinking is not bound by the laws of logic and reality. It is unlogical and permits the greatest contradictions with the outer world and in itself." From the examples given we can see how autistic thinking cut loose from logic will be directed; it has a tendency to represent desires as already fulfilled. "If we observe delusions in general we find that desires certainly play a great part in them, but that the delusion content is always dependent upon affectivity. The melancholic has depressive delusions and feels himself sinful, poor, incurably ill. The euphoric has grandiose ideas, he is a particularly clever man, and if his intelligence is somewhat disturbed, he may

possess the whole world and think himself equal with God." "The jolly do not think sad thoughts, the sad are even less inclined to let sad thoughts arise in them." "What keeps autistic thinking in the healthy person within certain limits is in fact logical thinking." "Where the balance between affectivity and logical thinking is upset, autistic thinking may get the upper hand." This occurs (1) in children, who lack sufficient experience "to discriminate logical possibilities." (2) "In subjects which are not sufficiently accessible to our knowledge and logic; in questions of the first principles, in the '*weltanschauung*' in religion, in love." (3) Where for any reason the emotions obtain too great a significance, as in strong affects, be they pathological or normal, whether they are caused by accidental circumstances or by the subject's own temperament. (4) "Where the connection of associations is loosened, as in dreams, in schizophrenia and so on." "While pleasant and unpleasant emotions have equal significance in the usual formation of delusions, in those emotions which are connected with particular ideas the effects of the two qualities are very different." There is a tendency to seize pleasure and repulse pain and ordinarily one will more easily call up pleasant than unpleasant ideas. Hence, "autistic thinking represents more fulfilled desires than realized fears." Suppression of unpleasant thoughts however does not always succeed and the repressed thoughts may "live on in the unconscious and from thence provoke abnormal symptoms which will be created in an autistic way," for instance someone wishes the death of a rival named Stout and goes in for a ridiculously exaggerated weight reduction cure to kill Mr. Stout in his own person.

Freud is inclined to identify what our author calls autistic thinking with the form of thinking of the unconscious. Autistic thinking may be just as conscious as logical thinking however, and every psychologist who does not deny the unconscious, knows that it also can think logically. Autistic thinking is peculiarly guided by such associations as correspond to the feeling-toned idea, that is to the desire or fear. Besides, secondary associations which in logical thinking are kept down as much as possible may become more prominent but only in so far as they can be used for the aims of autistic thinking. Thus objects spring from one object to a similar one and tend to generalize themselves. For example, a girl is frightened by a bull, the first impression being made by its genitals. She afterwards identifies these with the horns. Some years later she has forgotten the whole event, but becomes afraid of anything looking like horns so called by that name, for instance will not eat soup containing horn-shaped noodles. Symbolic images occur as well in the modern schizophrenic as in the makers of myths of old times and among savages. Hence some have spoken of an "archaistic form of thinking." The author cannot accept this opinion. Savages can think logically about things which they understand, but they understand fewer things, hence use more autistic thinking. Autistic thinking serves instincts especially in politics, particularly under the party system, which in many ways tends to lower public morality. How has autistic thinking served humanity? The boundary line between rational and autistic speculations cannot be laid down by human intellect. "What is inconceivable to-day may to-morrow become fact; what is firmly believed to-day may to-morrow become false." "Therefore, a humanity without autistic thinking could not have been developed." Nevertheless, both individual and national health need a balanced proportion between autistic and realistic, and the realistic must control the autistic.

8. *Personality and Psychosis*.—It is at times obvious that there is a relation between the mental characteristics of an individual and the type of the psychosis which he develops. Particularly is this noticeable in the manic-depressive psychosis, mild forms of which in particular sometimes appear to be but an exaggeration of the characteristics peculiar to the victim. The same thing is noticed to some extent for some paranoias, while in the more severe forms of manic-depressive the sequence is less clear. Coming to dementia præcox, the matter becomes more complicated.

Many authorities in fact regard this as distinctly a disease process of organic nature. Nevertheless there is a certain proportion of cases, though perhaps small, in which the patients have all their lives shown certain peculiar traits. In two series of studies the author found that in as high as from 50 to 60 per cent. of the cases the chief traits which had existed before the mental breakdown belonged to those which he at that time called "shut in tendencies." To these tendencies Bleuler has more recently given the name of "autism." He has also called attention to the possibility of a psychological interpretation of many of the symptoms and to many of the precipitating mental causes, for while physical causes play their part in precipitating attacks, mental causes are the more prevalent and important. These are not however always directly apparent, but often careful analysis is needed to elicit them. Comparing dementia præcox with the neuroses it is difficult to regard from the same aspect, the recoverable functional neuroses and dementia præcox, which leads to permanent mental deterioration. Nevertheless there is in both a defect in adaptation. There is no doubt that processes of an organic nature may, and do, accompany mental processes. Any defective endowment might hence be expected to have its organic basis.

Now the investigation of the brains of precocious demented sometimes reveals actual defects of development, such as atypical nerve cells, cells with two nuclei, indeed evidences of microgyria, etc. Again dementia præcox seems to be more often associated with congenital intellectual defects than manic depressive insanity or the neuroses.

Dementia præcox develops upon a more serious defect of constitution than, for instance, manic-depressive, though we cannot see any direct relation between intellectual and other constitutional defects. While dementia præcox is excluded from the so-called psychoses of the degenerate in the narrower sense, the author thinks that if we look without prejudice, transitions from dementia præcox to other constitutional disorders will be difficult to deny, although it cannot be proved clinically.

Considering manic-depressive insanity we find in a considerable proportion of the persons affected evidences of exaggerated emotions and a tendency of these emotions to be of the same nature as the psychosis and the parallelism between the personality and the psychosis is more evident than is generally believed. Manic-depressive insanity often represents an increase of a reaction type and its constitutional nature is generally admitted. The idea is frequently expressed that its attacks may represent an organic reaction and the fact that in general paresis and in Basedow's disease similar conditions exist, suggests this. On the other hand its symptomatology is essentially that of the normal emotional states and these latter are reactions to definite situations and determined by internal and external mental factors. Such a causal relation is often denied, however, in the case of pathological states. Interesting is the problem of anxiety. The anxiety found in neuroses has been looked upon as essentially an

organic reaction. The studies of Jones and Seif seem to the author to show that the genesis of such attacks of anxiety are in many cases explained by external and internal situations so that in pathological states, also, anxiety preserves its essential character as mental reaction. It is often impossible to find a precipitating mental cause in manic-depressive insanity. Nevertheless such causes may be found upon careful search more frequently than superficial investigation would lead one to think. It is to be hoped and expected that further studies will bring us more and more data showing the mental forces at work in this disease.

Both psychopathic personality and psychosis may be looked upon as determined by constitutional factors in the sense that the individual is found unfit to meet the demands made upon him either through inherent weakness or at times also through false attitudes developed through lack of training. In this last direction lies our hope of modifying these defects.

9. *The Personal Factor in Association Reactions.*—Purely psychological and quite technical. The author shows in general however that the reaction to the association experiment depends largely upon the personality of the subject, though the emotional state is also important. A study of the responses obtained shows that they may in general be divided into an egocentric and concrete category. A careful analysis of these responses with reaction times, etc., is capable of giving considerable information as to the personality of the subject.

10. *Neuropathic Inheritance.*—The author four years before, initiated a card system of the relatives who had been, or were in the London County Asylums, and had collected records of nearly 3,500 cases. It turned out that there were resident in these asylums 1,500 persons who were either related to one another or had relatives previously in the asylums, and that there were at the time 730 persons so closely related as parent and child or brother and sister in the asylums. Analyzing this material the author exposes his results in tables, by curves, etc. The following are some of the chief points brought out. In 3,485 cases made up from 1,620 families in which two or more members were insane, the statistics show (1) that among the insane offspring of insane parents daughters are more numerous than sons. (2) Among insane brothers and sisters, sisters predominate. With regard to "anticipation" or "antedating" he has found that there is a distinct tendency in the offspring of insane parents for the insanity to occur at an earlier age and in more intense form. In this connection it appears also that in the offspring of insane parents if insanity has not developed by the age of 25 years, the tendency for it to develop progressively decreases from this on. The author's tables show this very clearly.

Other facts also indicated are: (1) "The average age incidence of male and female general admissions for each decade is almost identical." The larger numbers of females in the asylums seems to be due to accumulation, the females having mainly less fatal forms of insanity, particularly less general paresis. (2) The female offspring of insane parents show a slightly greater incidence early in life than the male, the most marked difference being in the early involutional period, 35-44 years." (3) Taking the total offspring, there is far greater incidence in early life under 25 years. Of 663 offspring of insane parents examined, 95 or 1/7 were imbeciles. At this point several genealogical tables which illustrate very clearly the points made, are introduced.

A comparison of single with dual inheritance gave the following: In 25 families with insanity on the side of both parents, there were 154 chil-

dren. Of these 20 died young, 46 were insane, had nervous disease or committed suicide, and 88 were apparently normal. 34.3 per cent. of those reaching adult age were affected. In 193 families with single inheritance only, there were 689 children. Of these 52 died young, 43 were insane, etc., 594 apparently normal. 6.7 per cent. of those reaching adult age were affected. In comparing direct with collateral neuropathic inheritance, there were examined 63 families with direct inheritance, having 304 children. Of these 27 died young, 61 were insane, etc., 216 apparently normal. Of families in which there were insane aunts or uncles, there were 91, with 354 children. Of these 28 died young, 12 were insane, etc., 314 apparently normal.

The author discusses at some length the hereditary transmission of temperament and tendencies. In collecting his pedigrees, he noted frequent association of insanity or suicide "in a stock preceded by or associated with the existence of individuals possessing the melancholia, suspicious, brooding, self-centered, hypochondriacal temperament." It is not uncommon to find suicide in successive generations of such families. Associated with such temperamental evidences of degeneracy, epilepsy, alcoholism and the neuroses are not infrequent. Most interesting are his pedigrees which show the creation of neuropathic inheritance in an originally healthy stock by the marriage of cousins each of whom has a latent neuropathic taint or by the introduction of a positive taint through marriage with an unrelated person.

The author thinks that his pedigrees show that the idea held by a majority of practicing physicians "that a chronic blood poison (especially when occurring in successive generations) produced by the racial poisons, alcohol, syphilis and tuberculosis, can per se cause degeneracy in a healthy stock by a pathological mutation of the germ plasma, which can be transmitted," has a basis of truth despite the objection of the biologists, that the poison may be acting upon the individual by reviving a latent neuropathic trait.

11. *On the Etiology of Pellagra and Its Relation to Psychiatry.*—The author analyzes the various theories as to the origin of pellagra which he divides into the corn theory and the infection theory under the latter head being included infection by schizomycetes, by hyphomycetes, and by protozoa. None of these does he find proved. Nevertheless a prophylaxis based upon the idea that the disease is due to the use as food of spoiled corn has in Italy by improving the nutrition and the hygienic condition of the poorer part of the population decreased the disease to one third its former proportions. In psychiatry the problem has been to decide whether pellagra produces a specific form of psychosis, or whether it acts only as a determining cause of one of the familiar forms, particularly of melancholia and amentia. According to the author's view, the depression of pellagra is not to be identified with melancholia, nor is the confusion to be considered as amentia. As diagnostic points of difference he mentions that melancholia as a rule comes on comparatively suddenly in a patient who has generally had a similar period of disturbance before, while pellagra comes on slowly after a long period of symptoms which are improperly considered as neurasthenic, without emotional excitement. The mood in pellagra is particularly sad, though there are occasional flashes of what may be considered as "gallows humor ('Galgenhumor')." The mixed conditions of manic-depressive have never come under his observation. There is considerable inhibition in the pellagra patient so that he is difficult of access and often gives the impression of being confused or demented.

Delusions may occur but they are not fixed and the pellagra patient has always a clear realization of physical illness, and does not trace his sad mood to external causes like the melancholic but rather to his disease. Insomnia is less frequent in pellagra, and diarrhea is as frequent as constipation is in melancholia. The depression of pellagra does not pass into a manic condition but rather into one of confusion. This confusion reaches its most complete form in what is known as "pellagra-typhoid" the difficulty of diagnosing which from true typhoid and from other chronic and often obscure infections is often very great. A careful examination with the aid of all the resources of the laboratory may be necessary to protect us from error here. In these patients there are sometimes neurological symptoms which are foreign to amentia but belong to pellagra, such as hypertonia, and exaggeration of reflexes. The patients are really not so confused as they appear and can often inform us as to their personality, etc., though they are to a greater or less extent disoriented especially as to place. Realization of illness is always present. Hallucinations are rather infrequent and not so manifold as in amentia. These cases may sometimes resemble delirium tremens and senile or presenile, anxious states. In these cases the confusion is more like that of amentia, the motor excitement is pronounced and hallucinations are abundant. The question as to whether or not pellagra may directly produce dementia the author considers as unsettled. He does not think it probable but cannot deny that arteriosclerotic dementia may occur, the arteriosclerosis being at least indirectly due to the pellagra. Pellagra is not a hereditary disease and produces no hereditary psychoses.

12. *Psychic Disturbances Associated with Disorders of the Ductless Glands.*—Starting out with a plea for the closer coöperation between the other branches of medicine and surgery and psychiatry as well as among themselves, the author proceeds to discuss the mental state of a number of patients with disorders of ductless glands, especially with those of the hypophysis cerebri and the pineal gland, who have come under his observation during the past few years. It has long been recognized that giants are as a rule dull, if not of pathologically low intelligence. In acromegalics, recurrent periods of "feeling acromegalic" in which there is an intensification of hopelessness, lethargy and depression is more or less characteristic and has been graphically portrayed, by Dr. Leonard Mark himself a sufferer. However in only three of Dr. Cushing's series of 30 or 40 acromegalics was this condition pronounced enough to suggest a real psychosis. One woman committed suicide. In hypopituitarism adiposis and infantile sexual development may be the rule but some of the victims have for a time at least been quite bright, even precocious and may have been able to occupy for a period quite important positions. As a rule they are mostly placid and euphoric though the author has found some who were irritable and restless. He tells of some cases in which the administration of hypophysis extract, the implantation in the brain of a pituitary gland from a stillborn child, or partial removal of a hypophyseal tumor effected some temporary improvement. As evidence that reduced function of the pituitary gland may play a part in the phenomenon of hibernation, the author states that in a number of woodchucks in this state, the pituitary glands examined in his laboratory, showed the cells of the anterior lobe to be "small, undifferentiated and uniformly free from the granules and varied staining reactions which indicate stages of secretory activity." As to the possible transmissibility of an internal secretory derangement he thinks that there is some evidence, Halstead having found histological changes in

the thyroid glands of the puppies of a partially thyroidectomized bitch, while clinical examples are given in the author's monograph on the pituitary gland.

That precocious adolescence may occur in conjunction with lesions of the pineal gland is well known. The cases of two boys, in one of whom the secondary sexual characters had appeared at three and in the other at six years, are mentioned. Both of these showed mental deviation with lack of inhibition. In casting around for a pathological basis for the sexual deviations which play so large a part in the conceptions with regard to the psychoneuroses of the Freudian school, our author thinks that the internal secretions which seem to have so direct an effect upon the sexual activities may well be considered. This article contains much more that is of interest, but more especially from the standpoint of internal medicine.

13. *Primitive Mechanisms*.—A plea for the careful reconsideration and possible revision of our psychological conceptions and terms. The author is convinced of the necessity for studying all reactions as biological units, beginning with those which are most early manifested in the embryo and passing from the more primitive to the more complicated adjustments. "Adjustments taking place in isolated planes may be described in books but never occur in living things." "Since the synthesis of complex mental states brings us face to face with formidable theoretical difficulties, may we not avoid a dilemma by a more direct method of investigation of individual reactions?"

14. *Problems of Dementia (Demenzprobleme)*.—(In German, though a résumé in English is added.) The author as a former pupil of Wernicke was asked to choose some subject which should give an insight into the development of the school of thought which has borne the name of Wernicke, since his untimely death. Since Wernicke's work as a neurologist and especially his views upon aphasia have received such general recognition it is to-day considered so much a part of medicine that it cannot be claimed as a possession by any particular school. It is different with regard to his psychiatric views since no one, among his most devoted pupils even, has accepted them in toto. The genius of Wernicke and the influence of his ideas is however shown by the fact that while to practical psychiatry they do not appear at present to have contributed much, they have nevertheless considerably influenced psychiatric thought, while none of his pupils can be said to have added to them. As a personal scholar of Wernicke during the time he was preparing his "Grundriss der Psychiatrie," and on the ground of his own personal interest the author has chosen as the subject of his address the problem of dementia, since in no direction was Wernicke's interest so stimulating as in the study of defective conditions in the broad sense. It is generally conceded that the term dementia implies psychical inferiority though the question whether it must be permanent and irreparable or not, is still under discussion. For clinical purposes it is certainly desirable to distinguish between curable and incurable conditions, but how difficult this is, is shown by the fact that climacteric and some other depressive conditions in which a permanent defect has been assumed to be present, sometimes clear up. Such experiences however do not permit the assumption of a basic difference between temporary and permanent defective conditions in every case, and that the same cause may upon one occasion produce a temporary defect, but on acting further a permanent one is not excluded. This is particularly evident when we consider general paresis in its remissions and it is important to distinguish here between psychotic symptoms which in the narrower sense are capable of improvement, and

the loss of mental capacities which persist uninfluenced during the remissions. This brings up the question as to whether symptoms which are usually considered as part of a definite defective condition may not also occur, as transitory defects capable of repair. That this is possible the author thinks is proved by certain symptoms observed in presbyophrenia and in Korsakow's disease and more particularly by what is often observed in epilepsy. The same thing is illustrated in the somatic sphere by the occurrence after paretic seizures of paralytic phenomena which later clear up to a greater or less extent, but are rendered more and more permanent by successive attacks. The analogy between such purely somatic defects and the development of dementia, must ever awaken in us the hope of reaching a pathologico-anatomical conception of the psychoses, though so far we have made little progress in this direction. Still more difficult is the answer to the question what constitutes dementia, or can we consider dementia as identical in every case differing only quantitatively from case to case or from time to time in the same case. This last would seem to be denied in nearly all text books, since there are described for instance, paralytic, senile, epileptic, hebephrenic dementias, etc. We are however here describing the cause not the character of the dementias.

The problem of dementia is closely bound up with that of intelligence. Is this a unique phenomenon upon which a number of more special manifestations are dependent, or the sum of a number of components combined in varying manner? From experimental results the former view would seem the more probable. The high degree of correlation which they found between a number of relatively simple capabilities (among which learning by heart was not included), led Krueger and Spearman to conclude that they were influenced by some central factor. The capabilities tested by them, however, are far from constituting actual intelligence in the higher sense. In such experiments the psychological methods of testing intelligence have generally been used, and it seems that these do not necessarily carry out the end of the psychiatrist who desires to learn whether or not dementia is present. These two problems are by no means identical, since for instance there are certain capabilities whose presence by no means implies intelligence but which cannot be absent without there being mental defect. For instance, idiots often are observant though not intelligent, while complete absence of ability to notice ("Merkfähigkeit") what is going on necessarily implies decided mental defect. It seems on the whole that we are even less likely to arrive at a unique conception for dementia than for intelligence. Foerster and Gregor who have carried out upon paretics similar investigations to those of Krueger and Spearman in normal individuals, have been able to show—in so far as testing such things is possible in paretics—that between the processes which in normal persons are in correlation, there is similar connection, and that the functions so correlated are injured to an approximately equal degree by the process underlying the disease, and have come to the even more important result that a process outside this correlation, ability to learn, is not only affected in varying degree but also that the difference in individual paretics is very great, this sometimes suffering more, sometimes less than the above mentioned functions. If such great differences are found from person to person in a disease anatomically established, how much the more are they to be expected in conditions in which no such relation can be determined! At the start it was postulated that dementia was to be considered as constituted by symptoms of loss or defect, but how separate these from the psychotic symptoms proper or those characterizing the disease? Indeed

many of these psychotic symptoms (in the narrow sense) as affects, delusional interpretation of the examination, may make any proper testing for defects impossible.

Of much greater difficulty is the separation, theoretically so easy, of defect symptoms from psychotic symptoms in the restricted sense. The most typical example of this is the long-disputed point as to whether in the paranoiac, inability to correct delusional ideas, which to us seem entirely absurd, is due to the nature of the delusions themselves or is to be considered evidence of dementia. It appears to the author that such questions can never be solved as long as one clings to the conception of dementia as a unique process, since in spite of the examination of new material they are bound to be answered in the sense of one's individual ideas upon the subject. Similarly the question may be put, whether in the terminal stage of dementia præcox there is really a dementia in the restricted sense, or simply reduced ability. It has always been known that such patients know more than they appear to, and Penon has shown by his investigations upon old asylum inmates how moderate the actual defect is, since they not only know a great deal but can under the tests accomplish a great deal, when conditions are favorable, that is when those factors which disturb carrying out the tests or make them impossible and so give the impression of severe defect, can be eliminated. These influences are known to vary from minute to minute and to depend largely upon external influences, the personality of the observer, etc. All this shows that the appearance of dementia may be produced not only by actual loss of capacities, but also by other factors, as tension, negativism, etc., which are more nearly related to psychotic than to defect symptoms, and which show their irritative character by their sudden variations, etc. The idea of a unique dementia is further shattered by the difference of the defects found in paresis, in epilepsy, in presbyophrenia and in juvenile defective conditions, while there is always the question whether the defects demonstrated by our methods of testing really constitute the "essence" of dementia. Penon in his experiments above mentioned, found in an old "circular" much poorer response to his tests than in many precocious demented though this individual did not produce the impression of being anything near so demented as these. When it comes to be considered not only whether there is an actual central disturbance, but also not what dementia actually is, but what is to be understood by the term, we bid fair to lose ourselves in boundless discussion. Even defects in knowledge must be interpreted with much caution since the testing of recruits by Rodenwald, etc., has shown that the line between actual defect and mere ignorance is not a hard and fast one. Whether the Binet method which has proved so useful for testing school children is equally applicable for adults or suitable for testing for dementia is a question.

A question analogous to that of dementia is, what is disturbance of consciousness and what constitutes it? Considering loss of memory for a given period as proof that consciousness has been lost has, in the author's opinion led to unwarranted extension of the conception of epilepsy. Studies on epileptics, typhoid patients and cases of concussion of the brain show that loss of consciousness is made up of a number of components and that the study of these elementary symptoms does not yet make this phenomenon clear to us, as it leaves the feeling that we have still missed something, perhaps the most important thing. In so far as dementia presents itself to us as a unique whole, the idea that this impression is made up from the cumulative action of different components appears more satisfactory than that the individual symptoms are different results of a common basic disturb-

ance. As to the anatomical basis of dementia, the idea has obtained that forms of insanity leading to dementia must have an anatomical basis, and here it seemed that the bridge should come, over which we might hope to pass from organic brain disease to mental disease. These hopes seem at the present time little likely to be realized, though of late it has been asserted, perhaps with too great emphasis, that all psychoses with anatomical basis must lead to dementia while in all the others it is futile to search for pathologico-anatomical changes.

In the last decade, Nissl, Alzheimer and others, following in their steps, have furnished us with valuable data for the control on autopsy of our clinical diagnoses, especially by their contributions to the pathological anatomy of paresis, senile and arteriosclerotic alterations, Alzheimer's disease and idiocy, but these are important more from the neurological side, than as giving anatomical bases for sharply defined and readily diagnosable psychic disease pictures. We can hardly speak of any correspondence between anatomical findings and forms of dementia either quantitatively or qualitatively. It does not however seem preposterous to hope that with gradual improvement of technique we may enlarge our knowledge of the grosser changes in the brain and may bring them more and more into connection with disturbances of function which experience has taught us are connected with certain regions. We cannot, however, at present connect "psychological" defects with lesions of definite cortical cell layers.

On the whole it takes more optimism to speak of the possibility of an anatomical localization of mental diseases as brain diseases to-day than it did when Wernicke wrote 20 years ago, and the increased skepticism in this regard is to be referred, on the one hand, to the lack of fulfillment of former hopes, and, on the other, to the altered views as to the nature of the mental processes which have become more and more apparent of late in the last years.

15. *The Inter-relation of the Biogenetic Psychoses.*—The greatest amount of work in psychoanalysis has been done in the neuroses and important information as to their mechanism has been obtained. In the psychoses proper less has been accomplished, but still something has been done. For instance, it has been shown that in dementia præcox there are unconscious psychogenetic mechanisms akin to those found in the neuroses, and that there is a regression of mental processes toward a more infantile type, Bleuler's autistic thinking. Freud's studies in paranoia are also of interest as showing by comparison that paranoia, dementia paranoides and paraphrenia, represent stages in the regression of the mental processes toward a more primitive ontogenetic state. In studying a psychosis we have to consider not only the disease but also the patient's reaction to it and the conclusion is now justified that many of the symptoms are really defense mechanisms as in hysteria, for it is well known that symptoms usually considered as hysterical are found from time to time in the psychoses. The status of manic depressive insanity has been changing of late years, mainly growing at the expense of the dementia præcox group, the hard and fast boundaries which were formerly thought to exist between the two groups having been found to be non-demonstrable often. The author and Brill have found that there are cases clinically appearing as manic depressive insanity which are shown on psychoanalysis to be of the nature of anxiety hysteria. Though these cases should really be separated from the manic-depressive group, the author thinks that closer investigation will show as close relations between manic depressive and hysteria as between dementia præcox and hysteria. On the whole, he is coming around to the view that in

psychoses proper (insanities with organic basis being excluded from consideration), the symptoms observed depend not so much upon essential differences in nature, as upon different types of reaction to a fundamentally allied group of difficulties, namely "intrapsychic conflicts of a biological nature."

16. *Prognostic Principles in the Biogenetic Psychoses.*—On the basis of what he had observed in the clinic, Kraepelin taught that a sufficiently keen analysis of clinical pictures would permit the differentiation in the early stages of cases of good and of bad prognosis through the predominance of certain symptoms or their groups. That this is however not entirely safe or satisfactory is shown not only by the statistics of Kraepelin's own clinic, in which the diagnoses of dementia præcox have fallen from at one time as high as 52 per cent. of admissions to 7 per cent. in 1909, while they vary at other clinics from 30 per cent. at Zurich to 12 per cent. at Ward's Island. In opposition to Kraepelin's idea that a correct diagnosis settles the prognosis, the author has formulated a psychobiological interpretation of these psychoses, according to which the symptoms represent biological reactions of the mental type, the psychosis expressing a defective biological adaptation of the individual. This conception does away with the necessity for assuming that the outcome is determined by the kind or severity of a disease process itself unknown, but accounts for the course and termination by the action of the forces at work in the individual and the type of reaction itself, some reaction types being unfavorable for return to normal, others less so. The pure forms of manic-depressive reaction type represent chiefly an emotional deviation and the symptomatology is remarkably free from elements opposed to correction later, so that the prognosis is good.

In dementia præcox however, the psychosis appears to be the "outgrowth of a long-continued period of unhealthy biological adjustment on the part of the individual." Meyer showed clearly how indications pointing toward defective adaptation could often be recognized long before obvious breakdown, in many cases even in early life. It seems improbable that general prognostic principles such as brought forward by Kraepelin can ever be successfully applied to large groups of biogenetic psychoses. As a contribution to the prognostic value of a symptom group the author reports the result of his observations on the so-called catatonic syndrome. While originally considered as of very unfavorable significance, its importance has greatly diminished in this respect, since even Kraepelin admits that 15 per cent. of his catatonics recover and it is well recognized now that the syndrome may occur in a number of psychotic conditions both of organic and of functional origin. We have no satisfactory explanation of it, even psychoanalysis having failed to throw much light upon the subject. Negativistic stupor has however the general characteristics of a defense reaction or protective mechanism, whereby the individual shuts out the external world. Obstinacy and irritability may simulate negativism and where there is much perplexity or retardation the patient may appear negativistic.

The author has analyzed 80 cases from Ward's Island in which this syndrome was present and has divided them into three groups. (1) Catatonic Psychoses Associated with Deterioration. A study of these seems to show that the catatonic reaction was always preceded by a period of ominous prodromes which should give some hint as to the probable termination. (2) Psychoses Essentially Catatonic Throughout, but Ending in Recovery. Most of these were instances of catatonic stupor or depression with catatonic behavior. Many lasted over two years, and recovery was a

surprise. One of these was in a catatonic condition for over 5 years but ultimately recovered. There was nothing in her mental make-up or the development of the psychosis to suggest deterioration, but the symptom-picture and the long duration led to an erroneous prognosis. The author thinks that when the reaction is exclusively or chiefly stuporous the prognosis is good. Unfavorable signs are odd behavior, grimacing, stereotypy with gradual establishment of stupor, negativism and fixed postures. (3) Psychoses presenting Besides Catatonic Symptoms Manic Depressive Features. Many of these were wrongly judged to be deteriorating types because of overvaluation of the catatonic manifestations. There were instances where in the same individual there was at times a manic-depressive and again a catatonic syndrome, and it appeared that a catatonic phase might replace the depressive one in a circular case. There was sometimes a peculiar intermingling of the symptoms. In summing up the author concludes that from the catatonic syndrome alone, a correct forecast cannot be made. More reliable prognostic data are gained by a study of the constitutional tendencies and the mode of development of the psychosis. When the anamnesis reveals a gradual change in the personality with indications of that type of defective biological adaptation which we are coming to look upon as a foundation for dementia præcox, the prognosis is bad.

17. *Anatomical Borderline Between the So-called Syphilitic and Metasyphilitic* being more especially in the meninges, the metasyphilitic in the author's findings in six cases—illustrated by some excellent plates—which show how difficult it is to separate the changes of cerebral syphilis from those of general paresis. He thinks that the most helpful viewpoint is to regard syphilitic and metasyphilitic disorders as usually distinct varieties of the same general process. This viewpoint is based upon the following facts: In the brain stem and spinal cord of both groups, the character of the exudate and its distribution are much the same. In the cerebral hemispheres while the exudate is the same in kind, it differs in distribution, the syphilitic being more especially in the meninges, the metasyphilitic in the cortex as well, though there are borderline cases which may be interpreted either way. Further, gummata and syphilitic endarteritis sometimes co-exist with general paresis. In tabes with mental symptoms the brain may show changes either of syphilis or of general paresis. The serum reactions coincide. The discovery of the *Treponema pallidum* in all of these disorders points to a common origin. Nevertheless, it is desirable to keep them separate for study in what seems to be their natural relationships.

18. *Pernicious Anemia*.—A report of six cases of pernicious anemia associated with mental symptoms, with clinical histories and anatomical examination (with plates). Two cases were of depression, four of a paranoid condition in which the delusions developed upon a basis of irritability and suspicion. The author concludes that mental diseases of more than incidental importance may be associated with pernicious anemia, and that these are not always of the amentia type usually described, but are more chronic, showing resemblance to mental disorders symptomatic of toxic conditions. There may be pathological changes in the cortex of the brain, in their finer aspects like those observed in mental disorders associated with toxic influences and also specific lesions, which resemble those occurring in the spinal cord in pernicious anemia.

19. *Closing Remarks*.—A look over the field covered by the numerous excellent addresses with a retrospective glance at what has been accomplished by American psychiatry in the past twenty years and suggestions as to its tasks for the future.

C. L. ALLEN (Los Angeles).

The Journal OF Nervous and Mental Disease

An American Monthly Journal of Neurology and Psychiatry, Founded in 1874

Original Articles

REPORT OF SEVEN CASES OF BRAIN TUMOR

(WITH AUTOPSIES)

WITH ESPECIAL REFERENCE TO DIFFERENTIAL
DIAGNOSIS*

By S. P. GOODHART, M.D.

NEUROLOGIST OF THE MONTEFIORE HOME AND HOSPITAL; ASSISTANT PROFESSOR
OF CLINICAL NEUROLOGY, COLUMBIA UNIVERSITY, NEW YORK

AND

H. CLIMENKO, M.D.

CHIEF OF CLINIC, DEPARTMENT OF NERVOUS DISEASES, O. P. D. MT. SINAI
HOSPITAL, NEW YORK; ADJUNCT NEUROLOGIST OF MONTEFIORE
HOME AND HOSPITAL

FROM THE NEUROLOGICAL DEPARTMENT MONTEFIORE HOME AND HOSPITAL,
NEW YORK

The conception of neoplasms of the brain dates back to early medical annals, even to the time of Hippocrates; but in spite of decided progress, especially in recent years, in our knowledge of the physiology and anatomy of the nervous system, the recognition and localization of brain tumors too often to-day confront us as a perplexing problem.

* Pathological notes were made by Dr. David Felberbaum.

While there are really only two areas wherein pathological changes apparently fail to give us symptoms, namely the right frontal and right temporal regions, yet even lesions in areas of recognized functions are often overlooked or misinterpreted not infrequently by competent observers. Wherein lie the causes? Let us consider a few of these.

A commonly misleading factor is the distortion given by the functional element as in our cases S. L. and S. S. Both of these patients were unquestionably of an emotional type, inclined to hysterical symptoms, and on account of this a true organic lesion was long overlooked. Since we are by no means certain as to how long the tumor in this case may have been in process of development, with the possibility of remissions, it is not unlikely that the apparently purely functional symptoms were really due to the presence of the growth, especially because of its location in the frontal lobe.

Another common source of error in localization diagnosis is the overlooking or disregard of very early manifestations. The neurologist makes light, perhaps, of the first symptoms, or even fails to obtain an accurate account of the initial, often purely subjective manifestations; well versed, doubtless in brain anatomy, his deductions are sometimes made entirely upon objective and subjective findings at the time of the examination; there is overlooked the possibility, at least, of already dealing with indirect or distal symptoms. These may be evoked by secondary changes within the cranium, such as edema, dilatation of the ventricles, interference with vascular supply, forcing of contents of the posterior fossa in part into the foramen magnum, etc.

Two of our cases furnish excellent examples. In one (S. L.) an easily operable growth was overlooked in its location by reason of predominance of thalamic symptoms at the time the operation was determined upon. The then existing symptoms were really secondary, remote from the growth and due to indirect pressure. These thalamic symptoms developed late in the course of the disease. A careful analysis of the history shows that the initial symptoms pointed to the exact location of the growth.

In the case of A. S., in which the tumor was far anterior, affecting the frontal convolutions, the right cerebellum was found jammed into the foramen magnum which explained the presence of symptoms directly pointing to a lesion in the posterior fossa.

Further: strictly motor symptoms may be misleading. For example: localized true motor spasm, regarded as a result of cortical irritation—really cortical epilepsy—is doubtless often due to a stimulus from some remote part of the brain. Our case of S. S. illustrates this. The case was long regarded as one of epilepsy. The cortical motor area was not directly involved and the provocative element must be sought elsewhere. Thus while we know that increased cranial pressure may give rise to general convulsions, we are accustomed to look for the Jacksonian type—localized seizures—as starting from direct irritation in the motor cortical area. However, personal observation in our case cited, and others of a similar nature, lend strong evidence to our belief that irritation, especially by neoplasm of the subcortical motor fibers anywhere in the motor radiation, may cause localized seizures. As pointed out in one other of our cases (S. L.) and in accord with not infrequent observation, instead of the usual subtemporal or occipital decompression for the relief of intracranial tension and pressure, it would seem more advisable if the skull were opened at the probable, or at least at the possible site of the lesion.

Observation of the features in the various cases here reported are made under their respective histories. The usual symptoms met with in brain growths are fully dealt with in the special text books.

CASE 1.—Miss S. L., aged 37 at the onset of her illness in June, 1912, family and early personal history essentially negative, died March 31, 1914.

The earliest history bearing upon the illness which caused her death dates back to June, 1912. There was first noticed by the patient sensations in the fingers of the left hand which were doubtless paresthesia; there followed successively weakness of the left hand, fingers and arm, though the patient continued her work; soon, however, the finer movements, such as required in piano playing, sewing, etc., became impossible. At this time the diagnosis of neuritis was made by her attending physician.

In November, 1912, there was beginning weakness of the left lower extremity and also excessive emotional effects, variability of moods and uncontrollable laughing and crying. She then had no headaches, no nausea or vomiting, no vertigo, no eye symptoms; nothing to suggest general symptoms of neoplasm. During this month the patient suddenly lost all power of the left lower extremity and fell to the floor; this weakness disappeared within twenty-four hours. Doubtless because of the general nervous manifestations and what was then regarded as a purely functional

paralysis, the diagnosis of hysteria was made. The weakness of the left arm and leg advanced so that the patient dragged the left foot and the left arm was held in a position of semi-flexion due to contracture. At this time also the patient felt pain in the arm, but it was indefinite in character and not constant. Somewhat later an asymmetry in facial innervation was observed affecting especially the lower branches.

Dr. Abrahamson and Dr. Strauss saw the case at this time and after a thorough examination suggested the probability of a thalamic tumor. This diagnosis was certainly warranted by the thalamic symptoms. Status of the case by these gentlemen has been incorporated in our paper.

In March, 1913, at which time the patient's condition was as last described, a diagnosis of thalamic thrombosis was made. A blood Wassermann was negative.

In April, 1913, the patient began to complain of frontal headache, continuous in character and unrelieved by drugs. For some time previous to this there were convulsive attacks confined to the left arm, coming on about six times daily and characterized by a violent tremor of the left hand and forearm, which lasted several minutes and was followed by a spasm in which the hand was tightly clenched so the patient was unable to open it; this spasm lasted about a minute. Upon admission to Mt. Sinai Hospital during this month, physical examination showed left facial palsy, abdominal reflexes diminished, and the left arm held approximated to the side, there being only slight contractures. The left lower extremity motor power was only slightly diminished but the knee jerk was diminished. At this time an eye examination was made; both fundi showed evidence of beginning choked discs; the remains of a former choreo-retinitis were emphasized by the examiner as in character and position suggesting a syphilitic lesion. (See A. S.) Lumbar puncture and blood Wassermann gave negative results. There was also noticed at this time hyperesthesia over the left half of the body. A series of salvarsan injections was given and a gradual but progressive improvement of the power of the left arm and leg was noted.

Early in May, however, the involvement of the discs progressed to manifest optic neuritis of both eyes. There likewise developed a tremor of the *right hand, fingers and forearm* strongly suggesting the tremor of paralysis agitans; it could be influenced by the will though intensified on attempts at voluntary movement. In the latter part of May, following a number of salvarsan injections and large doses of iodides, the fundus examination showed marked improvement and the optic neuritis was reported in the regressive stage.

In June lumbar puncture and Wassermann again gave negative results; neosalvarsan was given again, however, and marked improvement in all symptoms was noticed. Deep injections of salicylate of mercury were also given.

In December, 1913, the right pupil was found larger than the left; corneal reflex absent on the right side. It was also found at this time that the paresis of the left facial was both for voluntary and involuntary innervation.

The history of the case for several months was not accessible and therefore cannot be included here. We know, however, that during this time there were attacks of projectile vomiting with frontal headaches.

In January, 1914, the following bedside notes were made: Left hemiplegia involving lower and middle facial; a fine, rapid tremor of the right hand; stereognostic, deep tactile and postural senses absent in left upper extremity; pain sense intact; left homonymous hemianopsia; upon smiling, left facial paresis disappears, but the left facial muscles move after the right innervation has begun.

At this time a diagnosis was made of deep-seated neoplasm involving the thalamus and tracts between the thalamus and the cortex. A decompression, or callosal puncture was advised. It is of importance to mention that in the latter part of January, 1914, some indefinite psychic symptoms were observed; whether based upon hallucinations or illusions the observations of that time fail to make clear. Contrary to the fact and without material basis for such belief, the patient claimed persons were coming in and leaving the room during the night and that she was being constantly removed from one place to another.

On January 2, 1914, a right subtemporal decompression was done; only slight pressure and normal pulsation were found. An exposure was also made for puncture of the corpus callosum. It was then supposed that only the outer table of the skull was penetrated; the hemorrhage immediately became so excessive that it was not deemed advisable to further expose the patient since the measure in this case seemed not so strongly indicated. This tumor might easily have been removed *en masse*.

On January 12, the patient's mental state was found to be so clouded that a careful psychological examination was impossible, though attempted, as the patient was not in condition to lend herself to analysis. The abdominals were still absent; the left hemiplegia with the usual contractures had advanced and become almost complete. The left knee jerk was still absent, with decided Babinski on the same side. The facial paresis was present both in voluntary effort at control and expressing emotion. The hemianopsia had disappeared. The pupils reacted fairly promptly, the left somewhat slower than the right. Tremor of the right hand was still present. The left corneal reflex diminished. This continued until death supervened on March 31, 1914.

The pathological findings as observed in the gross specimen are as follows: Viewed *en masse* from the right lateral aspect a large endothelioma is seen occupying a deeply excavated portion of the

right hemisphere at about the middle. The growth replaces fully a third of the external surface. Posterior to the growth the convolutions are found compressed so that actual loss of brain substance is only apparent and not real. The several convolutions can be easily identified though forced together by the compressing action of the growth. The tumor extends from just anterior to the ascending frontal convolution, exercising its greatest and direct pressure, therefore, at this part, cleaving the frontal convolution as the growth extends into the depths; below it just touches the first temporal convolution. The depression, or crater-like appearance of the center of the growth with its adherent dura and pigment is due to the trephining begun for the callosal puncture. The mesial aspect of the right hemisphere shows the tumor reaching to the corpus callosum. The tumor measures in its horizontal diameter 3 inches; antero-posteriorly, $2\frac{3}{4}$ inches and $2\frac{1}{8}$ inches in depth. These measurements were taken after hardening. The



FIG. 1. Miss S. L. Showing cavity in hemisphere left after growth was removed *en masse*.

tumor is easily separated from its bed, leaving the latter as a glazed surface showing numerous depressions corresponding to the cauliflower-like excrescences of the growth. A sagittal section through the center of the tumor shows the compressing effect on the deep structures especially the central ganglia, more particularly the optic thalamus of the same side. The internal capsule is entirely dis-

torted and displaced. The thalamus is flattened. The radiate fibers from the cortex, especially anteriorly, are greatly compressed.

Upon removal of the tumor from its bed and then looking upon the surface of the brain from above, one sees a decided enlargement in the lateral diameter of the right hemisphere, especially the anterior portion. Whether this is due, as claimed by Spiller and others, to a real hypertrophy of the brain, or rather to displacement, cannot be positively decided by macroscopic inspection. We are inclined to think that in this case the appearance of enlargement is really illusory.



FIG. 2. Miss S. L. Section of hemispheres (tumor removed) showing depth into which tumor descended into the brain.

[Path. Note. This growth shows the minute structure of a typical endothelioma. One sees spindle cells arranged in densely packed layers. (Same as Mrs. H. G.) The nuclei take on a deep stain while the protoplasm is not faintly stained. Few vessels are seen. No psammomatous material is found in the specimen.]

There are some interesting features in the analysis of this case. Firstly, it is indeed remarkable that a tumor of this size should give symptoms covering a period of 21 months only. It will be noted that the first symptoms were paresthesia of the fingers of the left hand. This symptom was a clue to the site of the growth and was due either to pressure on the cortex, or more probably on cortical thalamic fibers.

A rather unusual symptom suggesting the involvement of the frontal lobe and anterior thalamic region was the early dyspraxia noticed in lack of proper use of the fingers and hand, especially in the finer movements; the early diagnosis of neuritis from these symptoms shows the need of a more careful discrimination.

The course of the anatomical involvement of the extremities with the psychic symptoms, especially of an emotional character, likewise suggested the position of the tumor in the motor area extending into the frontal lobe. Though the tumor must have been of rapid growth, there was during the first year conspicuous absence of important general symptoms such as headache, vomiting, vertigo, or ocular symptoms.

The sudden loss of power in the left lower extremity, disappearing within 24 hours, was doubtless due to temporary edema or transitory circulatory disturbance. The diagnosis of an hysterical paralysis at this time before patient was admitted to hospital was doubtless based on the absence of all general organic symptoms of tumor, the undue emotion of the patient and transitory paralysis. These symptoms are readily explained by the anatomical position of the growth and we have here another example of the need of care in not mistaking organic for functional symptoms. The slowness of the onset with subjective sensory symptoms was due either to interference with the coronal radiation, as is evident from a glance at the specimen or, less likely but entirely possible, indirect pressure upon the internal capsule. Also, because of the spontaneous pain in the arm, pressure on the thalamus, or even a thalamic lesion was to be thought of; and this diagnosis was made somewhat later.

It is remarkable that only in April, 1913, late in the disease, there appeared frontal headache, continuous and unrelieved by drugs. The frontal headache as a late symptom could be accounted for on the ground that much pressure on the frontal lobe was not exerted and the meninges not extensively involved; in other words, the tumor probably grew out of the depths, beginning in one of the deep sulci, early exerting some pressure immediately in the region of the thalamus; the gross surface appearance justifies this conclusion.

The convulsive attacks confined to the left arm, followed by spasm but with no interference in consciousness, was strictly a focal symptom and again pointed to the localization of the growth. It is to be noted that the hemiplegia observed in April, 1913, was characterized by only slight contractures; this is explained by reference to the specimen which shows no real destruction of the motor fibers. A hemiplegia of this character could likewise be

explained as due to thalamic involvement, especially with the later development of hyperesthesia over the left half of the body.

A very interesting phenomenon was, on the hemiplegic side, diminished knee jerk with which there was later associated the extensor toe reflex, or Babinski sign. This is a rare association in organic growths and we believe may be explained as the result of interference with impulses passing along the spinal thalamic system. It cannot be explained by indirect pressure on the cerebellum, or by pressure on the spinal roots through internal hydrocephalus. A diminished knee jerk cannot in this case be explained by contractures of the opposing muscles, or by atrophy of the quadriceps extensor group. The explanation usually given for the absence or diminution of knee jerks in brain tumors, increase in pressure upon the spinal roots because of the increase of tension of the cerebro-spinal fluid, is not borne out by the absence of this phenomenon in the case of A. S. here reported in which the contents of the posterior fossa were in part forced into the foramen magnum by pressure.

The fact that improvement under salvarsan and mercury was for a time markedly progressive leading to a diagnosis of the probable specificity of the disease, shows the need of care in such conclusions. Repeated negative Wassermann blood and spinal fluid speak emphatically against syphilis. The development of a tremor of the right upper extremity, strongly suggesting the tremor of paralysis agitans and yet intensified on attempts at voluntary movement, spoke for a midbrain tumor. On the other hand, though not having the characteristics of frontal lobe tumor, it is barely possible, considering the anatomical location of the growth, that the frontal lobe was responsible for this motor symptom. There is, however, but little doubt but that it was due to midbrain pressure. Here again, like many other late manifestations, its value for localization is diminished and, as a symptom, misleading.

The diagnosis of a deep seated neoplasm involving the thalamus and tracts was certainly justified by the characteristic thalamic syndrome of left homonymous hemianopsia; fine, rapid tremor of the right hand, left hemiplegia, with left paresis in facial mimicry doubtless due to pressure on the pulvinar.

The later hallucinations or illusions, though transitory, were due to further involvement of, or pressure upon, the frontal lobes. When surgical relief is attempted in indefinite lesions, this is only

one of the many cases that illustrate the need of operating directly over the probable site of the tumor and of making careful examination of the brain surface and environment as far as possible including free use of the aspirating needle for finding pathological tissue. It was found post mortem that the operation for corpus callosum puncture was directly over the seat of the lesion and the growth might readily have been enucleated; on account of profuse bleeding, the investigation was not carried far enough since it was thought that the hemorrhage was of osseous origin as it was not known that the tumor was directly beneath.

CASE 2.—J. K., aged 49, first symptoms 1910, died Feb. 21, 1914, family and personal history unimportant. This case was in the service of Dr. Abrahamson to whom we are indebted for the privilege of utilizing the status made by him.

While eating a sour pickle patient was suddenly unable to swallow, coughed a few times and became unconscious for half an hour. Two weeks later there followed a paresis of the left lower limb. The next symptom developed a year later when the right leg was similarly affected and, shortly after, the left arm. There were also observed at this time some disturbances in respiration and in speech though the nature of this was not noted.

During the year preceding death, the symptoms were diplopia; headaches and paresthesia of the lower extremities; fibrillary twitches with crenation of tongue; frequent and annoying eructations, independent of food and in the nature of a spasmodic contraction; memory for past events defective; a series of clonic convulsions involving the left face, right upper and lower extremities without loss of consciousness; the lower limbs showed characteristic spasticity, double ankle and patella clonus with double Babinski and active abdominals; right pupil was irregular and reacted sluggishly; there was some ataxia in the left upper extremity and disturbance of postural and stereognostic sense.

Blood Wassermann was strongly positive. Lumbar puncture was absolutely refused by patient. Within a day or two before death patient became irrational. The eye grounds, even so late as a month previous to exitus, showed right fundus normal; in the left dilated veins and clouding of the disc. Death was due to sepsis.

The pathological nerve findings are as follows:

Skull.—In the median line, just anterior to the bregma, there is a spherical elevation about three inches in diameter causing a distinct prominence of the skull. The latter is very smooth in outline. The interior of the skull cap, corresponding to the area of bony enlargement, is much roughened and firmly adherent to the dura. This hypertrophy is not hard, can be easily incised to a depth of about $\frac{1}{4}$ inch on the inside and on the outer surface to

about $\frac{1}{8}$ inch depth. Between these two soft layers is solid bone.

Brain.—Outer surface of the dura corresponding to the bony condition described is roughened, thickened, jagged. The dura is adherent to the cortex of the brain over both hemispheres from



FIG. 3. J. K. Sections of hemispheres showing infiltrating endothelioma growing almost symmetrically.

the end of the superior frontal convolution almost to the occipital lobe posteriorly.

In the fresh specimen what now shows as a somewhat solid tumor mass really looked like a mass of infiltrating material extending almost in butterfly form symmetrically along the median line into the hemispheres, practically obliterating in part the longitudinal sinus. The tumor mass involves almost entirely the right parietal lobe, the two halves of the tumor fusing in the middle

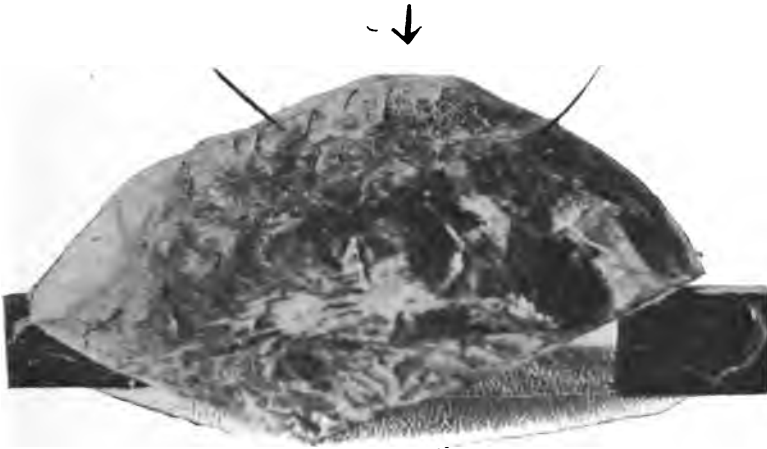


FIG. 4. J. K. Showing hypertrophic osteitis over the parietal bones at vertex.

line. The cortical leg centers on both sides are almost obliterated; posteriorly the growth has a distinctly infiltrating character and reaches almost to the corpus callosum. In the gross fresh specimen the tumor had somewhat the appearance of a large gumma, but shows microscopically to be an endothelioma.

In contrast to the specimen previously shown., also an endothelioma, this tumor could not have been excised completely.

[Path. Note. Histologically this tumor belongs to endotheliomata, but in many places the cells are massed together; all are cuboidal in shape; the nuclei show mitotic figures and there is an invasion of the dura and pia and the latter are very much thickened; throughout the growth are seen many arterioles which are very markedly sclerotic, some entirely obliterated by the endarteritis. At the periphery of the growth the brain substance is infiltrated with masses of endothelial cells. This is an endothelioma with malignant changes. There is also an endarteritis, probably of syphilitic origin.]

An interesting feature of this case lies in the fact that both the tumor of the brain and the osteitis of the skull entirely escaped clinical recognition.

Throughout the history of the case, two etiological factors were operative: one, the development of the neoplasm; the other the influence of the specific element. An analysis of the clinical history as compared with the pathological findings enables one to make some differentiation. The symptoms due to general specific involvement were especially those involving the basal nuclei; difficulty in swallowing, speech and respiration; fibrillary twitchings with crenation of the tongue; frequent and annoying eructations, doubtless due to spasmodic contraction of the diaphragm. Disturbances in the intellectual field, involving memory, with perhaps likewise the pupillary differences, may also be regarded as symptoms due to specific involvement of the nervous system.

The focalizing symptoms, those due to the neoplasm, were those involving the extremities, beginning with the paresis of the left lower limb and progressing in anatomical sequence. Of further interest was the absence of optic disc involvement, in spite of a neoplasm of such large size, until very late in the disease. In this case, as in the case of S. L. and in many reported by others, it is evident that one cannot be guided too closely by changes in the optic disc. Further, this case is analogous to the one just mentioned in that it shows that there may be large intracranial growth with but little that suggests the general symptoms of tumor. A glance at the specimen indicates how extensive may be the destruction of the longitudinal sinus without manifestations.

Just what influence there was upon the development of the growth from the presence of the specific element, one cannot de-

termine. The vessels in the growth itself, however, show a tendency to obliterative endarteritis. The decidedly malignant character is distinctly shown at its periphery. There is a peculiarly suggestive symmetry in the development of the growth with its butterfly wing form of extension on either side of the middle line; it may be largely due to the circulatory disturbances of a specific nature. The extensive infiltration precluded the possibility of operative relief.

In the gross specimen, the neoplasm might very easily have been mistaken for a large gumma. The pathological changes in the skull were of the nature of a hypertrophic osteitis. An interesting question is the relationship of the pathological changes in the skull to the intracranial lesion; the osseous changes were not distinctly of a specific character and it is not unlikely that the process was secondary stimulation in growth given to the cranial vault by an underlying neoplasm.

CASE 3.—S. S., aged 40, died Nov. 16, 1912.

Family history shows nothing of interest; personal history likewise. A relevant fact is the mental infirmity of two of the patient's children, one of whom is now at Randall's Island. The earliest history we have of the present illness dates back four years. The onset is described as a sudden, burning pain in the right upper extremity beginning in the tips of the fingers and extending upward as far as the shoulder. There were also "sticking pains" in the right side of the face; the mouth was pulled to the right side. The convulsive attacks always began in the thumb of the right hand. It was further observed that several minutes before the attack a peculiar sensation was felt in the anterior mid-parietal region. With this sensation as a warning, or aura, the patient would be enabled to seek a place of safety. He would then give a cry and go into general convulsions.

After the first attack he remained in bed several days and was attended by a physician. It is also recorded that the convulsive movements passed consecutively from the right thumb, to forearm and arm, face and right leg. In later attacks the left leg was included in the seizure and finally the left face. After the first attack the patient remained well for several weeks. The seizures coming on at first every two months steadily increased so that four years after the initial seizure the paroxysms averaged several daily. After the initial attack there was a transitory weakness in the right upper extremity. This weakness became pronounced about four years later and then retrogressed. Simultaneously there were marked cyanosis and edema of this extremity and this likewise was transitory.

A noticeable weakness of both legs several years subsequent to the onset of the symptoms showed a decidedly spontaneous improvement. The patient related that examinations for sensory disturbances made previous to 1910 showed no abnormalities. Likewise for some time previous to admission there were attacks of vertigo and headaches not localized. Beginning some four years after the initial symptoms there appeared clonic spasms of the left platysma and lower face, causing the drawing of the left side of the mouth and contractures of the muscles of the left side of the neck.

The patient relates that in 1909 his "right hand felt very nervous" as he expressed it; he could not bear to touch it, but felt no numbness and no tremor. The patient also described a peculiar, subjective feeling, referred to the eyes; it seemed to him as though his eyes were as "a cat's eyes in the dark." Shortly after admission to the hospital a number of epileptic seizures were observed. These were at times confined almost entirely to the right side, as previously described. This was followed by exaggerated deep reflexes and Babinski on the right side and ankle clonus. During the several months after admission to the Montefiore Home and Hospital, that is from June to October, 1910, convulsions, as described, were frequent. At this time the seizures were not preceded by sensory aura and invariably began in the thumb and second finger of the right hand to which at times the convulsive movement would be confined. There were likewise observed at this time occasional convulsive movements of the right eyelid; likewise spasmodic contractions of the left platysma and sternomastoid turning the head strongly to the right. It was seen that the convulsive paroxysm often terminated with contractures of the left platysma muscle. At times athetoid movements of the right upper extremity were observed.

In September, 1910, that is a few months after admission, the following notes were made: Of the cranial nerves, the first, third, fourth, fifth, sixth, eighth, ninth and tenth normal. There was a constant twitching of the sterno-cleido mastoid. There were constant convulsive movements of the left side of the face; the protruded tongue deviated to the right. The fundi of the eyes showed a suggestion of neuroretinitis. The abdominal, epigastric and cremasteric reflexes were lively on both sides. There was no Babinski, Oppenheim, Gordon, or Mendel sign. Right wrist jerk was exaggerated and there was markedly increased knee jerk with ankle clonus on the right side. There was no astereognosis. There was diminished power with tendency to contracture in right upper and lower extremities. What is described as "an intention tremor" was observed in the right hand. No sensory disturbances.

Notes made in November, 1910, by some observers resulted in the case being regarded as a functional one. It was presumed

that many of the symptoms were exaggerated and, in fact, feigned. Apparent confirmation was given this theory by very marked improvement in all the symptoms in January, 1911. The first suspicion that the case was purely of an organic nature was in the latter part of January, 1911. The great variability of the symptoms, affecting especially the spasticity of the right leg, and of the emotional variability in the patient which in itself seemed markedly to affect the intensity of the spasms of the muscles of the left side of the neck caused the erroneous conclusions.

In January, 1911, the case was regarded as one of probable gliosis of the brain. In April, 1912, mental symptoms were ob-



FIG. 5. S. S. Section showing gliosarcoma in the left hemisphere just under and involving the cortex. The cavity appearance is due to the breaking down of the tumor mass.

served, consisting particularly of confusion. At the same time there was observed also spasmodic twitchings of both upper lids in both facials. There was also a marked clonic spasm of the left lower facial, likewise rigidity and flexion at the elbow joint of the right arm and clonic spasms in both forearms and hands.

Patient died November 16, 1912, in general convulsions.

The pathological findings are as follows: Tumor is seen in the left hemisphere involving the cortex. Anteriorly it involves the second frontal convolution and is about one inch from the longitudinal fissure. Extending posteriorly it involves the cortex for about two inches just reaching the ascending frontal convolution. Further, it extends inferiorly for about two inches pressing upon the first temporal convolution. The growth likewise descends into the depths of the hemisphere infiltrating the whole substance and destroying the portion of the corona radiata fibers coming from frontal and in part also those from the ascending frontal and parietal areas.

The tumor in its posterior third becomes entirely subcortical, excavating the brain substance and leaving only a shell of cortex about one-eighth of an inch in thickness. Thus the tumor extends directly into the hemisphere for a distance of two inches. The excavated portion is oval in shape, its longer dimension being antero-posteriorly.

The dimensions of the growth are: anterior-posterior diameter $3\frac{7}{8}$ inches; lateral diameter 2 inches; vertical dimension $1\frac{1}{4}$ inch. [Path. Note.—This growth consists of very closely packed minute cells, having darkly stained nuclei. The cells infiltrate the brain substance. Cells can be seen having very much larger cell bodies than others and these show likewise multipolar processes stretching out between other adjacent cells. The growth is a gliosarcoma.]

Although the initial symptoms were of an epileptic nature and the case was regarded as such for some time, it is another example emphasizing the need of great care in making the diagnosis of a true epilepsy in a patient who has reached middle life without a history of previous attacks, though these may have been so far back as infancy.

The initial symptom was of a sensory character; that is, localized paresthesia, corresponding however to the primary motor involvement. The anatomical situation of the tumor is far anterior to the generally recognized seat of sensation in the cortical or subcortical region. This early sensory symptom, therefore, doubtless the result of direct irritation, speaks strongly for the presence of a certain degree of sense perception in the motor zone. This we believe is entirely in accord with the fact that a motor cell being stimulated by an afferent fiber must of itself be possessed of some sensory quality. This is further demonstrated by observations in the true epilepsies where sensory *aurae* sometimes precede motor symptoms and doubtless start from direct impulses in the motor area as in the case under discussion.

We frequently observe paresthesia and even pain immediately precede a convulsive attack of the painful member. The peculiar sensation of pain, felt by the patient just preceding the convulsive attack, was doubtless of a referred character and due to meningeal irritation. The sensation was very nearly over the seat of the tumor. This symptom having been present as a very early manifestation points to a cortical rather than subcortical starting point. It will be observed that later the growth descended into the depths. This further emphasizes the importance of percussion of the skull for tenderness. The fact that the initial seizures began in the area affected by the neoplasm and then became general, well illustrates the familiar observation that for diagnostic localization the initial member affected points to the seat of lesion. The edema and particularly the cyanosis in the right upper extremity, transitory and

occurring under these conditions, are strongly suggestive of an organic cerebral lesion and must be regarded as an important diagnostic sign.

The only general symptoms of neoplasm previous to admission to the hospital were paroxysms of headache and vertigo. These were not recognized at the time as associated with organic intracranial trouble.

A splendid example of Monakow's diaschisis is afforded by the involvement, as the tumor progressed, of the motor area on the side of the lesion; there were clonic-tonic spasms of the muscles of the left face and neck due to the irritation of the cortical representation of these muscles. Thus the stimulation passed by diaschisis to a corresponding localized area in the opposite hemisphere. Another symptom to be explained either by edema or diaschisis was the thalamic sign of the abnormal sensation felt by the patient in his right hand; he "could not bear to touch the hand; it felt very nervous." In conjunction with this symptom we have the associated thalamic sign, athetoid movements of the right upper extremity.

For several years following the initial symptoms, because of the associated functional manifestations due to an emotional temperament, the organic nature of the disease was masked. However, paroxysmal seizures of headache and vertigo, later with a low grade of neuro-retinitis, should surely have suggested organic disease. It behooves us once more to call attention to this point, namely that organic symptoms may easily be overshadowed by a variety of functional manifestations so frequently found in patients the subject of organic nervous disease. Psychic symptoms consisting essentially of confusion of ideas, a rare form of psychic manifestation in a lesion of this character and position, were observed as only transitory and very late in the progress of the disease. Doubtless true psychic symptoms may be expected only when both frontal lobes are involved.

CASE 4.—Miss A. S., aged 25, admitted to the Montefiore Home and Hospital April 4, 1911, died Sept. 28, 1913. This case was from the service of Dr. Abrahamson whose diagnosis in the case was correct. His status is utilized in our report. Family and personal history show no etiological bearing on the subject of our present study.

The first manifestations associated with the illness that caused her death appeared in 1909 as slight frontal headaches; these were

paroxysmal and became more intense. There followed short attacks of unconsciousness during which there were convulsive twitchings of the right facial muscles. These attacks were sometimes preceded by transitory blindness; almost invariably as a kind of aura, epigastric distress and vertigo. There were likewise occasional occipital headaches and transitory periods in which the gait was at times ataxic and sometimes staggering with a tendency to fall to the right. Early in the course of the illness there was sudden but temporary failure or dimness of vision. With the first year almost total blindness supervened. A conspicuous symptom rather early in the course of the disease process was attacks of extreme weariness and somnolence.

About a year after the initial symptoms a fundus examination disclosed marked papillitis with pronounced edema, especially of the right eye, and signs of beginning secondary atrophy in the left. The left pupil was large and not responsive; the right small with little or no reaction. An X-ray examination showed destruction of the sella turcica with probable separation of the skull sutures and rarefaction of the frontal bones—evidence of intracranial pressure. In the course of a few months subsequently there developed nystagmus in both external positions. Because of the rapid advancement in symptoms a subtemporal decompression was done at the Mt. Sinai Hospital about a year after the onset of the illness. On November 3, 1910, horseshoe incision was made, three inches from the base to apex, in the left temporal region. Bone flap was removed. There was much bulging of the brain but manifest pulsation. There followed hernia cerebri. Microscopical examination of the cortical tissue removed showed normal brain substance. Aspiration over the site of the field of operation, about 1½ years later, revealed the presence of cysts containing a clear, yellowish fluid. The latter congealed in a short time, forcing to the surface a thin fluid which boiled solid.

Olfactory nerve degeneration manifested itself by progressive anosmia, the sense of smell being more rapidly lost on the left side. During the course of this deterioration there developed symptoms similar to an acute coryza. Sight was reduced to differentiation between light and darkness, but this perception was finally confined to the nasal sides of the retinae. Over the trigeminal nerve distribution all forms of sensation remained intact and the corneal reflexes normal. There was later temporary paresthesia in the form of numbness over the left facial area. The third, fourth and sixth cranial nerves preserved their functions. At one time the left acoustic became temporarily involved; its affection was transient and not definitely ascertained.

In April, 1911, some two years after the onset of the illness, there was found complete optic atrophy without evidence of former choked disc. There developed also weakness of the right facial innervation and some ataxia of the right upper extremity.

The right abdominal reflex was less active than the left. A fine, rapid tremor developed in both hands. There were no astereognosis, adiadochokinesis, or apraxia. A marked psychic symptom, a pronounced euphoria and especially Witzelsucht, tendency to more or less constant drollery, developed during the second year of the disease. Intelligence remained absolutely preserved.

One night during September, 1911, the patient suddenly awoke with loud screams and calls for help. She stated that on attempting to turn from the left side, on which she usually slept, towards the right she suddenly felt her mouth drawn strongly to the right side. Her tongue was twitching and pulling in the same direction. The right arm became stiff and the entire right side was involved in strong clonic convulsive movements. The only medication given was a suppository of opium and though fearing a repetition of the attack the patient was free from symptoms of that kind on the day following.

There was very little change in the somatic symptoms of the patient except that her general nutrition became gradually poor. Some ten months before her death her mental condition began to



FIG. 6. Miss A. S. Section through frontal lobe showing large endo-thelioma beginning at the base.

show signs of deterioration. The hernial mass at the site of the operation for subtemporal decompression began gradually to increase in size. With rapid mental deterioration and constant loss of body weight the patient had a series of convulsive seizures with loss of consciousness, in one of which she died in September, 1913.

The cerebral hernia was found to be in part a thin cyst wall containing clear colorless fluid; the brain substance at the site of operation softened and cystic; the base of the brain flattened; right cerebellum jammed into the foramen magnum causing a depression in the brain substance. In front of the temporal lobe, at the base and extending from the chiasm forward there was a cauliflower-like growth about the size of a hen's egg, situated at the base of the brain, compressing and destroying both olfactory lobes

and optic nerves. The growth is symmetrically situated, its middle corresponding to the longitudinal groove, its anterior border extending $\frac{1}{4}$ of an inch beyond that of the brain itself.

Posteriorly it extends to a point corresponding to the Sylvian fissure. Its greatest width is at the level of the first frontal convolution on either side.

The growth measures about $2\frac{1}{2}$ inches antero-posteriorly and 3 inches laterally. It extends upwards, compresses and practically destroys the corpus callosum anteriorly. The tumor is well circumscribed and can be easily separated from the brain tissue proper. The important structures practically destroyed by the tumor comprise: (1) Olfactory lobes and tracts, (2) Optic nerves and chiasm, (3) Pituitary body, (4) Frontal convolutions on both sides and some of the fibers of the corona radiata, (5) Anterior portion of the corpus callosum. Oculo-motor nerves of both sides and also the crura cerebri show macroscopically the effects of pressure.

The initial symptoms point most definitely to the localization of the growth. It is not unlikely that this case is one of neoplasm originating in the sheath of the optic nerve. The paroxysmal frontal headaches with somnolence and transitory blindness, the somewhat later involvement of both olfactory nerves, should have immediately suggested the true localization. It is interesting to observe that the X-ray examination, while showing destruction of the sella turcica, awakened the suspicion that the growth might be a hypophysis tumor. A further suggestion of the pituitary as the site of the lesion was given by the nature of the retinal field picture since the perception of light was finally confined to the nasal sides. X-ray findings in pituitary growths must be guardedly interpreted. Corresponding to the irritative symptom, that is papillitis from involvement of the optic nerves (through pressure, probably, on the nerve sheath) there was the interesting analogous signs of irritation of the olfactory in the forms of symptoms almost identical with an acute coryza.

With the upward extension of the growth into the frontal lobes the characteristic Witzelsucht developed. In spite of the initial symptoms pointing distinctly to anterior brain involvement at the base, the transitory ataxia, with a tendency to fall to the right, likewise the occipital headaches, suggest of course posterior fossa involvement. As Bruns has pointed out, however, we may have a typical frontal lobe ataxia; this symptom having occurred early in the disease was probably not due to the jamming of the cere-

bellum into the foramen magnum. It is probable that this latter condition was due to the formation of cysts at the site of the decompression. In either case it is evident that for this process of posterior fossa contents being forced into the foramen, it is not necessary that there be a very great increase in intracranial pressure.

It is remarkable likewise that with this involvement of cerebellar tissue by direct pressure, really no symptoms pointing to these parts obtrusively developed.

CASE 5.—I. S. From the service of Dr. Abrahamson whose notes of the case we are permitted to utilize. The growth was correctly localized. Admitted to Montefiore Home and Hospital March 10, 1902. Family history gives but one relevant factor; the death of the patient's father of tuberculosis. His early personal history affords no important data.

For some years prior to the development of his intracranial symptoms he suffered from extensive pulmonary disease of a tuberculous nature and from about the same time also from severe attacks of migraine of right hemicranium, the paroxysms occurring about once in two weeks. A year after the beginning of the migraine attacks the right eye in toto showed inflammatory stasis doubtless due to post orbital venous congestion. This was followed by a gradual diminution of sight, the eyeball protruding progressively from the orbit. The pain about the eyeball and its protrusion became so pronounced as to necessitate enucleation, after which the acute symptoms subsided until within a few weeks preceding death. The only other symptoms recorded suggesting an intracranial growth were occasional general epileptiform seizures observed for a period of a few months preceding death. Circumstances made it unfortunately impossible to secure a complete history of this case.

The intracranial pathological condition as observed post mortem is as follows:

The skull cap is thin; its interior smooth. Convolutions of the brain are much flattened. The right side of the anterior fossa shows a large white endotheliomatous growth evidently having origin from the posterior portion of the optic plate, growing directly into the brain substance, involving the right inferior frontal convolution, the right olfactory bulb and the chiasm.

The dilation of the right lateral ventricle is enormous and is seen to compress the lateral surface of the frontal and anterior portion of the parietal lobe so as to cause a bulging of the surface across the middle line, distorting the opposite or left hemisphere. The temporo-sphenoidal lobe is compressed and markedly thin so that the ventricular wall reaches the cortical layer of gray matter. The central ganglia are pressed upon and distorted toward the left.

Looked at from above, the right hemisphere in its anterior half appears enlarged as compared to the left hemisphere. It is likely that this apparent enlargement is due to the immense hydrocephalus of the left side. This one-sided hydrocephalus can only be explained on the assumption that the opening from the third ventricle into the left lateral ventricle was closed by the compressing influence either of the tumor or of the change caused by the un-



FIG. 7. I. S. Section showing dilatation of right ventricle.

equal amount of fluid in the two ventricles. It is possible that there was a unilateral inflammatory process which closed the foramen of Monro on the left side. This growth presents a classical picture of psammoma. Whorls of spindle cells are arranged around a central psammoma.

It is remarkable that in spite of this large growth at the base and of the enormous dilatation of the lateral ventricle exerting direct pressure on important brain structures, the neurological symptoms were by no means of an obtrusive character. Indeed, intracranial lesion was not suspected although the case had been under observation by competent internists. Although not examined by a neurologist, one would surely have expected emphatic clinical evidence of a brain lesion in a case in which the intracranial pathological findings were so extensive. It is worthy of note, therefore, that clinical manifestations were not present.

Nevertheless, the case emphasizes the need of early, careful, neurological status; further, the need of an awakening suspicion of organic changes in some part of the nervous system, most likely frontal, in a case of intense migraine and intractable loss of vision, especially beginning after middle life.

CASE 6.—D. L., admitted to Montefiore Home and Hospital December 12, 1913.

The history is interesting in showing the direct relationship between extensive involvement of the lungs with tubercular disease and the later development of laryngeal tuberculosis with final tubercles in the brain. The earliest symptom we have of brain lesion was severe occipital pain antedating by about four years the development of intense vertigo on change from horizontal to a vertical position. There were likewise occasional attacks of nausea and headache. Vertigo, the most prominent symptom, was in this case characterized by its presence and intensity only during change of position. Finally, associated with the vertigo, were attacks of perspiration and prostration.

Macroscopically the brain findings were as follows: Marked congestion of the dura with cerebral veins greatly engorged. There was some edema of the pia. The lateral ventricles were much dilated and filled with fluid. There was found in the right lateral ventricle anteriorly a tubercular nodule of the size of a large pea immediately under the ependyma. The base of the brain was

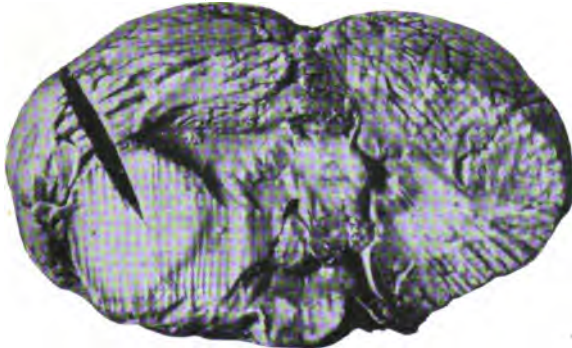


FIG. 8. D. L. Section through the cerebellum showing large tuberculoma in the right cerebellar hemisphere. Black cleft through growth is artefact.

normal. Both lateral lobes of the cerebellum showed semilunar depressions from having been jammed into the foramen magnum. In the right cerebellar hemisphere a large, hard growth was found about the size of a large pigeon's egg. Histologically, the neoplasm is a typical tuberculoma with central caseation, peripheral round cells and giant cells. There is no meningitic process about the mass.

CASE 7.—Mrs. H. G., aged 65, died October 27, 1911. Personal and family history have no bearing on the neurological findings.

The interest in this case lies in the pathological finding, post

mortem, of a tumor of the left frontal lobe extending upward from the base; it is an endothelioma, size of a pigeon's egg. The presence of the tumor was never suspected having given rise to no subjective complaints though the growth pressed both upon the left olfactory and the left optic nerves; macroscopically one sees that the left optic nerve is of far lesser diameter than the right. There is an area of softening involving the left internal capsule which accounts for the sudden hemiplegia of the right side which came on instantly with loss of consciousness three months previous to admission to the Montefiore Hospital.

Repeated neurological examinations did not point to intracranial growth. The hemiplegic syndrome was of course due to the vascular lesion within the internal capsule. Examination of the cranial nerves as made at the time was negative. The bedside record shows a careful examination of all the cranial nerves, but no mention is made of involvement of either of the olfactory nerves. The left eyeball had been enucleated, previous to admission, hence of course the very important symptoms both of the peripheral organ and the optic nerve itself were not obtained for record. In all probability the enucleation was due to ocular symptoms not recognized as of intracranial origin. That the olfactory involvement did not betray itself to clinical observation probably was due to the uncertainty of interpretation of such an isolated symptom as a unilateral anosmia. It is probable that the tumor had its inception in the optic nerve sheath and that the enucleation of the eye was done because of the protrusion of the eyeball. Tumors of the optic nerve are rare and doubtless easily overlooked.

[Path. Note. The brain in toto was remarkably small; indeed, appeared microcephalic. The convolutions, however, showed no atrophy; the diminutive proportions could not be ascribed to atrophic senile changes.]

The growth consists of spindle cells arranged concentrically, densely packed together. The nuclei stain intensely, the protoplasm faintly. There are very few vessels and these are dilated. Occasionally throughout the specimen psammoma material is seen. This picture is that of a classical endothelioma. To Drs. Abrahamson, Strauss and Hunt we beg to acknowledge our thanks for the privilege of utilizing some of their material.

COMBINED PSYCHOSES

By JOHN E. LIND, M.D.

GOVERNMENT HOSPITAL FOR THE INSANE, WASHINGTON, D. C.

A review of the literature of combined psychoses for the past thirty years reveals a surprising paucity of material. I have only been able to find a scant twenty-seven articles bearing on the subject, and some of these only deal with it indirectly. Here and there in the standard works on psychiatry are found allusions to the possibility of the existence of combinations, but only incidentally as it were. I shall not attempt to review or abstract this literature at any length for the number of case histories which I wish to touch upon will fill all the available space. Suffice it to say that it seems to be the opinion of psychiatrists in general that combined psychoses are fairly frequent, far more so than would appear by the published reports of such instances. Nosological fashions have been so mutable in the past few decades that it is often hard to tell whether a writer is referring to a different psychosis from the one which the reader had in mind or is merely using a different name. Something of the difficulty which attends the investigation of this subject even in the records of a single hospital will be mentioned below.

Appended to this article will be found a bibliography as nearly complete as I have been able to make it; I shall only mention briefly a few of the contributions most accessible to American readers. C. M. Campbell,⁸ writing in 1882, describes twenty-two cases of typhoid fever occurring in insane patients, all women. Unfortunately he does not give a full enough clinical history in any case to warrant a diagnosis of infection-exhaustion psychosis engrafted on the preexisting condition. Such a state of affairs may be suspected, however, from the statement, "The mental symptoms were intensified during the first few days of the fever." H. R. Stedman,²² writing in 1883, presents four cases which he calls instances of "change in the form of insanity during acute attacks." The first two cases in the light of our present day viewpoint appear to be manic-depressive psychoses in which the depressed

phase occurred first and was followed shortly by a manic episode. The third case was probably catatonic præcox and the fourth seems to have been a manic with excitements of varying degrees. R. P. Smith,¹⁹ in 1887, reports six cases of typhoid fever in insane patients. Two of these cases were apparently manic and the onset of the illness was marked by a subsidence of the manic symptoms and the appearance of delirium.

J. W. Stevens,²⁰ in 1907, has shown most indubitably a case of Korsakoff's syndrome coexisting with the depressed phase of manic-depressive psychosis and a constitutional psychopathy underlying both these. Krafft-Ebing speaks of general paresis developing in paranoiacs. Of course here we stumble on the vexed question of what constitutes paranoia, and if we hold to the old "true paranoia" of Kraepelin, and insist on both the clinical and serological proofs of paresis, it is extremely doubtful whether any such case has been observed or not; certainly I have found none reported in the literature, I recall none in my own experience and inquiry among psychiatrists of much larger experience than my own gives the same results. In 1911 Drs. Karpas and Poule²¹ presented a case of dementia præcox with tabes to the New York Neurological Society for differential diagnosis from general paresis of tabetic type. In the discussion it was stated by Dr. Kirby that no such combination had ever been observed at the Manhattan State Hospital.

Oberndorf,¹⁴ in 1912, describes psychoses occurring in constitutional inferiors and states that they all present anomalous features. He describes excitements and depressions, alcoholic episodes and præcox syndromes. Of course, strictly speaking, these cannot be called combined psychoses, unless we use the term psychosis in its broadest application, *i. e.*, the expression of an individual's abnormal reaction to a group of circumstances. Constitutional inferiority, psychopathy and other defective states are accompanied by excitements and depressions, especially the former and alcoholic episodes are to be expected, alcohol being the natural defense reaction in these conditions.

W. F. Lorenz,¹² in 1911, published a very good case of manic-depressive psychosis, infection with syphilis, and later on general paresis. Karl Graeter⁸ in 1909 published a monograph dealing with the inter-relations of alcohol and dementia præcox in which are cases of alcoholic psychoses on a præcox foundation, and L.

L. Smith¹⁰ found a number of such cases in investigating the cases of dementia præcox admitted to the Government Hospital for the Insane from the United States Army.

G. W. Gorrill,⁷ in February, 1914, published in the New York State Hospital Bulletin five excellent cases of combined psychoses. His paper is the most noteworthy clinical contribution to the literature I have been able to find. His first case was a dementia præcox of thirty-three years' duration, who became infected with syphilis when fifty-one years old and about six years later developed general paresis, as evidenced by the clinical, neurological and serological findings. His second case was general paresis developing at the age of forty-four in a woman who had had two attacks of manic-depressive type, one at thirty-three and one at forty-one. His third case was a manic-depressive with a history of three attacks, one at nineteen, one at twenty-two and one at thirty-four, followed by general paresis at fifty-seven. Autopsy showed a paretic brain and arteriosclerosis. His fourth case was not so clearly defined: A man of forty-three years contracted syphilis at twenty-nine, developed an attack of acute excitement at thirty-seven from which he recovered in nine months and seemed well for over five years when he suddenly became excited again and showed a manic-depressive reaction with the physical signs and laboratory findings of general paresis. It is possible that his first attack may have been paresis, followed by a five-year remission of symptoms; the only information given about the physical signs in the first attack is that the reflexes were exaggerated, his pupils reacted sluggishly to both light and accommodation and there was some tremor of tongue and fingers. The fifth case was a woman who had manic-depressive psychosis, one attack at twenty-six, another at forty-four, then at fifty-three an infection-exhaustion psychosis. There appears to be no doubt about this diagnosis.

The views of present-day writers in regard to combined psychoses are only found here and there, expressed in the course of articles dealing primarily with other subjects. In an article in the *JOURNAL OF NERVOUS AND MENTAL DISEASE*, in 1906, Dr. W. A. White, referring to clinical types, says, "They are not clean-cut entities, but are only groups of symptoms which either seem to occur more frequently in combinations or else have been more definitely and clearly seen because of the nature of their combinations. . . .

The great mass of cases seen are in combinations more or less intermediate in character. . . . There are combinations . . . which are quite frequent and which have only received inadequate notice and even for the most part go unrecognized."²⁶ In another place in the same article: "Because a person has manic-depressive insanity is no reason he should be immune from the ordinary diseases that affect the brain and impair the mind. . . . Thus we find the paranoia syndrome in manic-depressive insanity, dementia præcox and paresis; flight of ideas in dementia præcox; the Korsakoff syndrome in senescence and paresis; the presbyophrenic syndrome in paresis; katatonic rigidity and negativism in the toxic-exhaustive psychoses, and so on indefinitely."

Dr. E. E. Southard* says in regard to the question of combined psychoses, "I would say on theoretical grounds what I suppose most any of us would say, namely, that the postulate of combined psychoses may often be found to depend upon the *priori* selection of certain phenomena as constituting an entity, and then finding that entity alongside others in a different case. This, of course, would not constitute a logically rigorous proof of combination of psychoses. On the whole, however, having abundant faith in the future of our science and the confidence that there are multiple causes which may bring about psychopathic phenomena, I must say that I believe that the future will produce good and convincing proofs of the combination of psychoses."

Looking at the subject in the abstract light of speculation we may make the generalization that any two psychoses may occur in the lifetime of an individual. For instance, a man may have an episode of katatonia and recover, a few years later he may have delirium tremens following prolonged alcoholism, he may recover from this and be placed in an environment new to him and at the same time meet with sexual difficulties from which he develops a paranoid state with recovery. He may have typhoid fever and an infection-exhaustion psychosis and lastly he may round out his psychotic history by arteriosclerotic dementia and general paresis. All this is of course merely an imaginative exercise, but as a matter of fact there seems no reason why a toxic psychosis, for example, should not develop in an imbecile, a præcox, a manic or a paranoiac. Or, supposing any one of these four types becomes infected with syphilis, is there any reason why he should not de-

* Extract from a letter to the writer

velop general paresis or cerebral lues? Arteriosclerotic or senile changes are without doubt present in a great many cases of imbecility and *præcox* which spend their lives in institutions and these terminal psychoses are not recognized clinically. The notes on such cases toward the end are to the effect that they "show much dilapidation" or are "deteriorating rapidly" and it is only post mortem that the superimposed conditions are recognized. There is too great a tendency, also, in taking anamneses of new admissions to take it for granted that if the patient relates a previous attack it is similar to the then existing one. Closer examination of the patient or application to his family, his physician or the hospital in which he was formerly treated may bring to light a totally different psychosis.

The generalization made above, like most generalizations, requires modifying. It is dependent on the assumption that a psychosis appears as the result of a certain group of psychic and physical factors acting upon an individual and if we suppose that these groups vary in properties and potentialities at different times we can suppose them producing different psychoses. On the other hand we must take into consideration the fact that the individual begins life with a certain biological legacy, he is a phylogenetic endproduct. Starting then with this distinctively individual endowment, he is acted upon by a constantly changing environment, the change being always in the direction of complexity. The first change is from the all pervading warmth and support of intra-uterine life, unconditioned omnipotence, to the bright light and (comparative) cold of the outside world, from the complete fulfillment of all nutritive needs without conscious effort to the necessity for systematic and repeated exertions to secure the same result. From this first change the individual runs the gamut of complex situations until we find him an adult, coming into contact at a thousand points with his social heritage, a complex and exacting civilization.

We shall suppose that he adapts himself to the various requirements of life, moulding his environment here and there to suit certain individual psychic needs and occupies his niche in the social and business world with the minimum of friction. We have then an individual at least so far normal that he harmonizes with the general social scheme. Suppose, however, he fails to adapt himself when an exacting period of his life, such as the onset of

puberty, the first sexual experience, the first conflict with the business world or any other trying epoch calls for a readjustment of his scheme of things. The expression of his failure to react adequately we will call a psychosis and the symptomatology of this will depend upon his individual psychology, his make-up. Then, suppose him to have recovered from this psychosis, he again meets with a group of circumstances which call for extra psychic effort and he is again unequal to the situation. His expression of his failure would naturally take the same form, assuming of course the premise that the fundamental features of individual psychology are unalterable.

It is small wonder then that two such opposed methods of handling a situation as we find in the manic and in the *præcox* do not seem to occur in the same individual. In one case the libido, meeting with a bar to its progress, flows back upon itself and we have the reversion to earlier forms, the reanimation of old channels and the fixation at some point normally abandoned for good. This regression of the libido gives to the psychology of dementia *præcox* a peculiar aspect, the *præcox* lives within his self-created world which is admirably adapted to his psychic needs. He lies behind his self-erected fortifications and we can only surmise what activities take place there. The manic, however, pours his libido freely upon the outside world to divert attention from his psychic conflict, just as the garrison of a beleaguered fortress, threatened within by famine and mutiny, make sallies upon the besieging army. These antithetical modes of handling the difficulty are mutually exclusive and appear to be dependent on biological traits which are not altered to any extent during an individual's lifetime.

It will be interesting to observe whether or not the psychoanalytic method will make a change in this state of affairs. It does not seem impossible that some such state of affairs as this may obtain in the future. An individual may develop a *præcox* episode early in life and become aware of the source of his difficulty through psychoanalysis. His conflict being thus elevated to the surface of consciousness he may handle it successfully and take his place in the outside world. Meeting with a new difficulty years later he may develop a manic reaction, and so on.

The differential diagnosis between the dementia *præcox* and the manic-depressive psychoses has received its tons of printed trib-

ute to which I shall not add here. Enough to say that we have in the manic a quantitative deviation from normal and in the præcox a qualitative one. Hence it is that the præcox gives us a sense of strangeness while we find it easier to understand the manic's reaction. We can, so to speak, put ourselves in his place. On cross section there are cases which resemble both, but the longer they are observed the more readily they separate out. I have not been able to find dementia præcox and manic-depressive occurring in the lifetime of one individual.

The most common combination of psychoses appears to be an alcoholic psychosis engrafted on a præcox make-up. In fact this combination is so frequently seen that a natural doubt arises as to whether these are in reality alcoholic psychoses or whether they are not rather præcox episodes precipitated by alcohol. It is quite a common thing for instance in the Government Hospital for the Insane to receive a recently enlisted soldier diagnosed as an alcoholic psychosis or as a præcox caused by alcohol and find that the man is a præcox, probably of years' duration. The requirements of military discipline being too irksome for him, he took refuge in alcohol which accentuated the residuals of his old psychosis. I expect shortly to publish a paper dealing with this subject more fully. The relations between the two conditions are undoubtedly very intimate and it is questionable whether an alcoholic psychosis ever develops on a normal basis.

In investigating the subject of combined psychoses the records of the Government Hospital for the Insane from its establishment up to August 1, 1914, were reviewed, a total of 21,350 cases. From this number all those which had been readmitted to the hospital were extracted and only these were considered. This method is of course open to the objection that it does not cover cases which may have been treated in other hospitals for other psychoses or which may have passed through attacks not necessitating commitment. Such cases, however, would have to depend for diagnosis on descriptions given by the patient himself, often absolutely unreliable, the diagnosis made by a family physician, often unskilled in psychiatry or the routine method of examination and diagnosis in another hospital, often differing widely from the one used later on and dependent upon personal equations unknown to the writer. In going over the files 2,031 readmissions were found. Of this number a great many did not have sufficient clinical his-

tory during their earlier admissions to warrant a diagnosis. The staff in those days was not ample enough to permit of periodical notes on patients and such terms as "acute mania," "chronic mania," "acute dementia," "chronic dementia" and "acute melancholia" were in common use, terms which, needless to say, must be regarded as ambiguous now. When those cases from which no justifiable conclusions could be drawn as to their previous mental status had been weeded out there remained 808 cases with adequate histories of two or more admissions. These were all examined carefully. Many cases were thrown out which might perhaps without stretching the verities too far have been called combined psychoses, notably instances of præcox developing on a defective basis or alcoholic psychosis on a præcox basis. A large number of the cases consisted, of course, of attacks of manic-depressive psychoses. When the negative and doubtful cases were sifted out there remained 36 cases of combined psychoses and 5 cases of manic-like episodes in præcokes. These 41 cases may be divided as follows:

ASSOCIATED WITH DEMENTIA PRÆCOX

Alcoholic psychosis	6
Imbecility	3
Arteriosclerotic dementia	2
Infection-exhaustion psychosis	2
Hysteria	1
Total	14

ASSOCIATED WITH MANIC-DEPRESSIVE

Senile dementia	4
Alcoholic psychosis	2
Arteriosclerotic dementia	2
Infection-exhaustion psychosis	1
Total	9

OTHER COMBINATIONS

Psychopathic character and prison psychosis	2
Arteriosclerotic dementia and infection-exhaustion psychosis	2
Senile dementia and alcoholic psychosis	1
Senile dementia and infection-exhaustion psychosis	1
Psychopathic character and alcoholic psychosis	1
Arteriosclerotic dementia and alcoholic psychosis	1
Prison psychosis and cerebral lues	1
Paranoid state and general paresis	1
Imbecility and arteriosclerotic dementia	1
Infection-exhaustion psychosis and paranoid state	1
Paranoia and senile dementia	1
Total	13
Cases resembling præcox and manic-depressive	5
Grand Total	41

Abstracts of all these 41 histories would occupy too much space so where there exists more than one similar combination I shall only abstract one case, the most clearly defined one, and mention the others only by number. The number used refers to the records of the Government Hospital for the Insane, cases being numbered in the order of their admission. The first group of cases to be considered is the one comprising combinations of alcoholic psychosis with dementia præcox.

CASE No. 15983.—A colored male, aged 40 years when admitted to the hospital May 26, 1906. He was first admitted to the hospital on October 12, 1901, and discharged May 30 of the following year as recovered from a psychosis due to alcoholism. The medical certificate which accompanied him on his second admission stated that the present attack began in May, 1906, by erratic conduct, noisy at night, carried stones in his pockets, manifested destructive tendencies and delusions of a mixed nature. Thought that by throwing money about the floor he could remove sins. Physical examination on admission was negative. Mentally clouding of consciousness was present, he was elated and restless, spoke of having talked with the Lord who told him to clean up this place from top to bottom, he saw the Lord at night and the Lord appeared to him in a vision. Ward notes for several days following his admission, described him as highly nervous, excited and restless. After two months he became quiet and worked out about the grounds. He remained in this condition six years and when examined in October, 1912, he was found emotionally indifferent, not caring whether he stayed in the hospital or went home to live with his family who were anxious to receive him. His memory was uncertain and apperception and retention decidedly impaired.

The above seems to have been a case of two alcoholic psychoses occurring in a simple præcox. Other cases of the combination of dementia præcox and alcoholic psychosis were Nos. 20453, 19783, 16187, 19633 and 20562.

CASE No. 16878.—A white female aged 33 on admission, November 7, 1907. Patient was always considered weak-minded and did not progress normally at school. When about 28 years of age she complained of men coming in a balloon to her room at night and molesting her. She would scream and cry and run out on the street. She was admitted here for the first time September 24, 1902, and discharged 16 days later as recovered. Soon after leaving she developed delusions that certain persons were casting spells over her and hypnotizing her and was readmitted October 1, 1904. Mental examination at that time showed auditory and visual hallucinations, delusions of persecution and feelings of influence. She improved somewhat and was discharged July 1, 1905. When readmitted in 1907 she showed no hallucinations or delusions, but a general mental reduction and periodic attacks of

excitement. She remained in this condition and was finally discharged to the care of her family February 23, 1911.

The above appears to be a case of two *præcox* episodes occurring in a defective, probably an imbecile. On her third and last admission, the diagnosis seems to be imbecility alone. Other cases of *præcox* episodes occurring in imbeciles are Nos. 19128 and 18879.

CASE No. 4926 shows arteriosclerotic changes in a *præcox*. This patient, a white female, was admitted to the hospital June 3, 1880, being then 27 years of age. No systematic examination of the case was made until December, 1903, when the physical examination was negative. Mental examination at that time showed partial clouding of consciousness, auditory and tactile hallucinations and delusions of persecution. A diagnosis of dementia *præcox* was made. During the last year of her life she deteriorated rapidly and sclerotic changes were noticed. In July, 1911, a cerebral hemorrhage occurred and she died the same day.

Another case of arteriosclerotic dementia in a *præcox* is Case No. 21028.

CASE No. 15250 shows an infection-exhaustion psychosis followed in four years by a *præcox* which may or may not have existed at the time of the first admission. The patient was a colored female, admitted first September 3, 1904, suffering from a psychosis following ill health and childbirth. The diagnosis recorded then was acute confusional insanity and she was discharged as recovered on October 28 of the same year. She was readmitted on April 4, 1905; the mental examination then showed that she was confused, disoriented, talked incoherently to herself, had hallucinations of hearing and feared some one was going to harm her. She was indifferent to her surroundings, cross and irritable at times, memory defect, no insight, and reasoning and judgment were impaired. A diagnosis of dementia *præcox* was made; the autopsy four years later showed considerable shrinkage of the brain with no gross lesions. Another instance of this same combination is Case No. 19521.

CASE No. 18531 shows a hysteriform attack in a dementia *præcox*. The patient, a white female, showed her first signs of mental trouble in March, 1908, being at that time 27 years old. She obtained a revolver and attempted to shoot herself, broke a window and tried to cut her throat, also attempted to strangle herself with a handkerchief. She was admitted to the hospital on March 8, 1908. Mental examination showed apprehension, negativism, restlessness, clouding of consciousness, depression and visual and auditory hallucinations. She gradually improved and was discharged as recovered from dementia *præcox* on January 29, 1909. May 8, 1910, she suffered from a fainting attack and fell out of her chair. Following this she had several fainting attacks and appeared to be excited. She then became depressed and despon-

dent, complained of her tongue being thick and of inability to swallow, cried frequently without reason and took no interest in her household or her child. She was readmitted to the hospital on May 21, 1910, and showed no mental symptoms whatever, being discharged three weeks later as not insane.

CASE No. 16411 shows a manic-depressive case, complicated by senile changes. The patient was a colored female, aged 70 on her last admission, February 19, 1907. She had been a patient five times between 1895 and 1903, each time suffering from an excited phase of manic-depressive psychosis, with recovery. On her last admission she showed arteriosclerotic changes, was excited and remained so for several months. When she quieted down, she was found to have marked mental deterioration. From then on until her death in 1909 periods of excitement alternated with quiet intervals and during the latter she showed senile defects. Other cases showing senile dementia in manic-depressive types are Nos. 15399, 16168 and 15981.

CASE No. 19843 shows three attacks of manic-depressive psychosis, followed four years after the last one by an alcoholic psychosis. Patient is a white female, born in 1870. In 1900 she was treated in this hospital for one month, the diagnosis then being acute mania, discharged recovered. April 4, 1908, she was readmitted; mental examination showed her to be depressed and apprehensive. Consciousness was clear and she showed no delusions or hallucinations. She was discharged by the court nineteen days later and a diagnosis of manic-depressive insanity was made. May 5, 1908, she was readmitted with the same diagnosis and discharged four months later. In January, 1912, she began to drink heavily, was frequently intoxicated and on April 4 of that year she became voluble, resistive, removed her clothing and later became violent and entertained delusions of persecution directed against her relatives. She was readmitted to the hospital April 11 and was noisy, restless and confused. She continued in this state of confusion two days. At that time she had hallucinations of sight and hearing, imagined she saw animals and vermin on the wall of her room and would strike at imaginary objects on the wall. She remained in this condition about one week, when she became quieter, the hallucinations disappeared and mental examination revealed no psychotic symptoms. This is undoubtedly a case of delirium tremens occurring in a manic-depressive individual. Another example is Case No. 19641.

CASE No. 6729 shows a manic-depressive individual who later on developed dementia of an arteriosclerotic type, this diagnosis being confirmed by the autopsy findings. Patient was a white male, first admitted to the hospital in 1881 and again in 1886. Before this he had been treated in the Utica State Hospital. His age at his first admission was 41. The chief mental symptoms are given as depression with delusions of persecution, alternating

with periods of restlessness and exaltation. From 1902 on he demented and died in July, 1908, at the age of 68. Autopsy showed atrophy of the brain, opacity of pia and arteriosclerotic softenings. Case No. 20884 shows a similar combination.

CASE No. 20253 shows three attacks of what was probably manic-depressive psychosis, complicated in each instance by the excessive use of drugs and in the last one by an infection-exhaustion psychosis. The patient is a white female, born in 1868. In 1905 she was placed in a private sanitarium where she remained for nine months. Her mental condition while there is not known. After leaving she became addicted to the patent medicine habit and took vast quantities of these. In the summer of 1908 she attempted suicide on three occasions by taking laudanum. October 21, 1908, she was admitted to this hospital. Mental examination showed emotional depression, agitation and feeling of unreality; she thought her food was poisoned and that she had no bowels. This was succeeded by an excitement during which she broke dishes, threatened to kill herself, refused food and had to be tube-fed. She gradually improved and obtained good insight. On March 31, 1909, her case was considered at a conference of the medical staff and she was discharged recovered, April 3, 1909. She got along well, until July, 1910, when she began taking laudanum for pain in her side. She also took whiskey in large doses. She was readmitted to the hospital July 12, 1911, very much depressed, and discharged as recovered September 19, 1911. She got along well until the first part of September, 1912, when she began scolding her husband and nailing the doors so he could not enter the house. She would not allow anyone in the house and was active and talkative, working continually, canning and preserving until late in the night. She threatened to kill her husband with an ax, and would do nothing he requested of her. About this time, she developed a malarial attack and took a considerable amount of quinine. Following this, she had auditory hallucinations which excited her so it was necessary to commit her to this hospital. On admission she was quite disturbed, ran constantly about the dormitory, overturned the other patients' beds, talked to imaginary voices, approached the windows and called to the people passing by to come in and protect her. She continued this excited condition for about three weeks and then began gradually to improve. Mental examination at this time showed a flight of ideas, vague persecutory delusions concerning her husband, emotional depression but no clouding of consciousness or hallucinations. She was discharged as recovered on February 12, 1913.

A number of cases of the combination of prison psychosis with psychopathic constitution might be cited, in fact, it would seem to be the case that usually only those constitutionally predisposed,

develop such episodes. I have selected only two cases, and will quote briefly from one of them.

CASE No. 19440, white male born in 1885. The account of his school life was obtained from the patient himself, who stated that he was a bright scholar of unusual intellectual attainments. After leaving school he lost several situations on account of outbursts of temper; resulting in fights with other employees. He had several gonorrheal infections, the first one at the age of fifteen; was infected with syphilis at a very early age; used alcoholics to excess, and was intoxicated on numerous occasions. In the summer of 1909, he was arrested for robbery and released on bond which he forfeited by leaving the jurisdiction. When the police went to a nearby city to arrest him, he met them with a loaded pistol and it was necessary to use force to subdue him. At that time he was living on the earnings of a professional prostitute to whom he claimed to have been married for several years. Correspondence between him and this woman showed that he fully sanctioned her mode of life. He received a five years' sentence in the penitentiary, and soon after arrival there was noted as being excitable and irritable. He had several attacks described as maniacal, and during these he frequently attacked the attendant. He was transferred to this hospital April 7, 1911. On admission he was nervous and apprehensive, easily became excited, and there was clouding of consciousness. When an attempt was made to examine him the following day, he became intensely excited, profane and threatening. This excitement somewhat subsided and he was allowed the privilege of walking in the yard. He comported himself well, except when spoken to by the physician, when he would become quite excited. He made a plot to escape which was detected. In consequence of this, his privileges were removed, and this was followed by another excited attack. He was finally discharged as recovered from prison psychosis, August 10, 1911. A similar case is No. 19438.

CASE No. 19243 shows an infection-exhaustion psychosis followed several years later by an arteriosclerotic excitement. Patient is a colored female, and was first admitted to the hospital in 1908, being at that time fifty-four years old. She was diagnosed as an infection-exhaustion psychosis and discharged as recovered three months later. She was readmitted June 19, 1911. Mental examination at that time showed her to be extremely excited, yelling, singing, praying and running about, overturning the furniture. There was complete clouding of consciousness, memory was fair and the special tests were well performed. She believed that she could converse with spirits, and had the gift of prophecy. She thought the world was coming to an end and that she had been sent to warn the people. The physical examination showed sclerosis of the arteries and high blood pressure. She continued excited for several months, then quieted down. The

field of consciousness became clear and no hallucinations or delusions could be elicited. A diagnosis of excitement associated with arteriosclerosis was made, and she was discharged recovered. At the time of her first admission, she was suffering from septicemia due to a large abscess on the leg and lymphangitis. A somewhat similar case is No. 19623.

CASE No. 18556 shows a white male who was admitted to the hospital on February, 1907, suffering from alcoholic psychosis from which he recovered and was discharged in November, of the same year. May 30, 1910, he was readmitted and found to be suffering from senile dementia. He remained in the hospital until his death, March 6, 1911. Case No. 16300 shows a white female, seventy-eight years old who was admitted first in January, 1906, suffering from senile dementia, and was discharged to the care of her daughter in August of the same year. Five months later, she was readmitted suffering from an acute confusional state which was found to be due to kidney and heart disease, and cleared up under appropriate treatment, leaving the senile dementia as before. Case No. 21348 shows a psychopathic character, a colored female who has been admitted to the hospital on six occasions, suffering from alcoholic psychoses. Case No. 20872 shows a white male who was admitted to the hospital in 1898 and discharged a few months later. Readmitted in January, 1899, and discharged December, 1905. Readmitted June, 1907, and discharged November, 1907. Readmitted December, 1907, discharged January, 1910. Readmitted April, 1910, and discharged October, 1911. Readmitted February, 1913, discharged May, of the same year. On each of these admissions, he was suffering from alcoholic psychosis. He was readmitted the last time in August, 1913, suffering from excitement associated with arteriosclerosis.

CASE No. 14933 is a colored male, aged nineteen on his first admission in March, 1902. He had previously been an inmate of the district jail, being sent there for attempting to kill a man with a knife. While at the jail, he became violent, fighting the guards and beating his head against the wall. About two weeks after admission to this hospital he became quite excited while at dinner one day, jumped on one of the attendants and began to fight. He became involved in several other fights with the attendants during his residence here, but finally became free from excitement and was discharged in December, 1902, as recovered from prison psychosis. On September 22, 1904, he was readmitted and found to be disoriented in all spheres. He could give little account of the events leading up to his second admission. Up to February, 1907, he is described as being stubborn, resistive, excitable and had to be restrained on many occasions. February 2, 1907, he had a general convulsion accompanied by loss of consciousness and vomiting. Since that time, convulsions were frequent. He was unable to get about and was almost constantly noisy, shouting, yelling and

screaming. He was filthy in his habits and totally oblivious to his surroundings. He died June 14, 1911. Autopsy showed the presence of cerebral syphilis.

CASE No. 17465 is that of a white male who had a paranoid state when fifty-five years old, lasting about one year, and terminating in recovery followed five years later by general paresis which ended in death fifteen months later. At the time of his paranoid state, he was an inmate of the Soldiers' Home, and suffered from delusions of persecution and auditory hallucinations; at times he was so violent and assaulted the other members of the home on such slight provocation, that it was necessary to seclude him. An autopsy was performed at this hospital which confirmed the diagnosis of general paresis.

CASE No. 14618 is that of a colored male who was first admitted to the hospital in 1900. His age at that time was uncertain, being probably somewhere between twenty-five and thirty-five. He was found to be an imbecile and was discharged to the care of his family two months later. Two years later, he was admitted again and remained in the hospital seven months. Two years following this, he was readmitted suffering from excitement with delusions of a grandiose and persecutory tendency. He died in February, 1913. The post-mortem examination showed arteriosclerosis of the brain.

CASE No. 20661 shows a white male aged twenty-six on his first admission, January, 1909, when he was suffering from an infection-exhaustion psychosis following malaria. He was discharged as recovered in December of the same year. He was readmitted two years later with the same diagnosis, and was discharged as recovered at the end of seven months. He entered the Soldiers' Home in March, 1913, and was admitted here in May of the same year, with a history that he had various delusions, was noisy, required to be restrained and was destructive. Physical examination made here was negative. Mental examination showed him to be accurately oriented. No hallucinations could be obtained. He suffered from paranoid ideas directed toward the officers of the Soldiers' Home, who he thought discriminated against him. He soon recovered from these ideas, was given parole of the grounds and finally discharged November 3, 1913, as recovered from paranoid state. This is indubitably an individual of unstable organization who had two attacks of infection-exhaustion psychosis, and later on paranoid state.

The only case of paranoia complicated by any other psychosis which I have been able to find does not lend itself very readily to abstraction. The patient was first admitted to this hospital on April 6, 1910, Case No. 18451. He was then sixty-seven years old. At that time, his case attracted considerable newspaper attention. He was well known throughout Virginia, District of Columbia and Maryland by the legal profession. It seems

he began many years ago, to file suit against residents of these states, some of the defendants being his most intimate friends and neighbors. The courts all over these states were appealed to by this patient for redress for his fancied grievances. He succeeded in obtaining a number of judgments, and frequently attempted to execute these, the exact number of suits which he has filed is not known, but they are estimated as several thousand. His claim against one express company alone amounted to more than one million dollars. He was referred by the newspapers as the "King of Litigants," and the "Eternal Litigant." Lawyers refer to his case as "The Romance of the Law," and some of his cases are quoted in legal text-books in the states in which he operated. His case is undoubtedly litigious paranoia. Since being an inmate of this hospital, he has shown considerable deterioration, so that his case might be taken for one of senile dementia by any one not familiar with his history. He is quite childish in his appearance in conversation and his productions now are quite puerile. It is evident that in his present state, he would not be able to carry on his former activities. His defective judgment is shown by his transparent ruses to gain his release; for instance, when one of the physicians is away for a few days, he will hand his substitute a document which he says was written by the other physician. This is something to the effect that he is sane. He always states that the other doctor gave it to him before he left, in spite of the fact that it is in the patient's own handwriting. He writes sometimes to his relatives requesting that they bring him a revolver and some ammunition so that he might shoot the physician and gain his release. He writes a letter one week alluding to a certain lawyer in town as a "shyster" and a "crook," and a week later writes him to come out and handle his case.

A careful search of the records failed to reveal any cases of manic-depressive psychosis and dementia præcox occurring in the same individual, but there were five well marked cases of the manic-depressive reaction in a præcox case. One of these will suffice for illustration. Case No. 16984, male, white, age twenty-six on admission, single, letter-carrier, resident of the district. Father and brother were at one time insane. Early history unknown. Patient had been erratic in conduct for some time before his first admission here. July 1, 1905, he packed his trunk, left the city without notice, visited his relatives at Port Deposit, Md., acted strangely, and had to be brought back. He showed psychomotor activity, marked flight of ideas and emotional excitement. On his first admission on July 19, 1905, he was quiet and orderly. Improvement led to his being given parole on August 9, 1905, but three days later, he eloped and on his return after three days, he was erratic, talkative, noisy, fault-finding, disorderly. He was discharged by court, August 18, 1905. The patient then worked as a letter-carrier, and baggage agent. On several occasions when

seen by physician, there was mild flight of ideas, lack of insight, and evidence of excessive alcoholic indulgence. He sent irrational postcards to hospital employees. Medical certificate from Washington Asylum Hospital shows that since discharge by court, patient had been addicted to alcoholic excesses, and for three months prior to the last (second) admission, he showed pressure of activity, flight of ideas and emotional exaltation, with a tendency to probable impulsive attacks, and fits of violent temper, in which he was destructive, noisy, and profane. There were mild transient delusions of persecution, he imagined his food was being poisoned, and the police were after him. On readmission, January 17, 1908, there was psychomotor activity and flight of ideas, with some apparent looseness in the train of thought, and neologism. Stenogram four days after readmission shows a lessening of both psychomotor activity and emotional exaltation, with flight of ideas and distractibility. August 30, 1908, patient presented a dull expression, slowness of comprehension and ideation; disorientation for time and person, and partially for place; well marked indifference, disagreeable auditory hallucinations, lack of insight. On November 16, 1908, there was apparent mutism and negativism, even to refusal of food, so that tube-feeding had to be resorted to. Two days later, patient became a little more communicative and complained that the milk and eggs made him sick. On examination by the physician he was put to bed, a double lobar pneumonia developed, and on November 28, 1908, he died from cardiac failure. There was no autopsy.

The other cases which show this same combination were Nos. 17484, 18325, 19630 and 16776.

The above cases are not sufficient material upon which to base any generalization. A few points only appear somewhat remarkable and may perhaps be noted as affording basis for future investigation. It seems rather strange, for instance, that there were more psychoses associated with dementia præcox than with manic-depressive psychosis. Also it might have been expected that more than two cases would have been found of syphilitic psychosis engrafted upon another variety. The total number of combined psychoses found—forty-one—is rather small, when we consider the large number of readmissions to the hospital, but this is largely due to the inadequate notes made on cases up until comparatively recent years. Probably the future will uncover a larger proportion of combined psychoses owing to the fact that it is the present custom and has been for the past eight or ten years to obtain elaborate anamneses and to make copious notes at regular intervals.

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Society Proceedings

NEW YORK NEUROLOGICAL SOCIETY

**STATED MEETING, HELD AT THE N. Y. ACADEMY OF MEDICINE,
DECEMBER 1, 1914**

The Vice-President, DR. E. G. ZABRISKIE in the Chair

BRAIN WITH DOUBLE FRONTAL ABSCESSSES

By L. Casamajor, M.D.

The history was given by Dr. E. J. Smith, of Larchmont, N. Y. The patient was a mechanic and inventor, 50 years old, whose family history was negative. Between the ages of 20 and 30 he had suffered from several severe attacks of articular rheumatism, with heart complications. The patient had always been very active as a young man, and at one time was a long-distance bicycle rider. Until about twenty years ago he used alcohol habitually and to excess, but for fifteen years preceding his death he had been a total abstainer. He smoked constantly and used chewing tobacco daily. During the past ten years he had worked very hard at his trade and had accomplished a great deal in a mechanical line. He took no recreation excepting a few days each summer, when he did some yacht racing.

The exact date of the onset of his present illness could not be fixed. He was in the habit of receiving osteopathic treatment for minor ailments, and was under the care of an osteopath when he came under Dr. Smith's observation on March 10, 1914. At that time the patient complained only of a slight frontal headache and of being unusually tired. He had the appearance of being slightly jaundiced. The pupils were contracted, reacting slowly. The tongue was heavily coated and the breath offensive. The temperature was normal: pulse, 60 per minute and full. On his first day in the hospital he refused to stay in bed; he wished to go to business and insisted on smoking his pipe. On the following day he kept his bed and abstained from tobacco. He remained in his usual jovial mood and his symptoms were such as one would expect in a case of catarrhal jaundice. His condition remained practically unchanged for four days, without improvement in appetite, appearance of tongue, etc., in spite of active purgation.

On the fifth day it was observed that he was quite indifferent to his surroundings and very talkative. His headache had disappeared almost entirely and he still had nothing to complain of. When left alone he talked and whistled, and was not at all disturbed when he soiled the bed by an involuntary defecation. His pulse remained slow, and his rectal temperature on the fifth day was 101°. The urine was stained deeply with bile; it contained a trace of albumin; no sugar; there was a marked indican reaction and a few hyaline casts. No complete blood count was made, but a differential count showed 79 per cent. polynuclears. The stools were of-

fensive and lighter in color than normal. The pupils remained contracted and sluggish, acting equally. The ears showed nothing abnormal and there was no nasal discharge.

The patient remained in practically the same condition for another twenty-four hours, appearing indifferent when he was alone, but acting quite rationally upon being questioned. There was no paralysis and the reflexes were normal, excepting the pupillary. His temperature, on the evening of the sixth day, was 101° in the rectum; pulse, 54; respirations, 18. At 8 A.M. on the seventh day after his admission he suddenly developed a chill, his rectal temperature rose to 106° , he became comatose, developed Cheyne-Stokes respiration and died in three hours.

Two days prior to this patient's death his family was advised that the symptoms pointed to some unusual brain lesion, the exact nature of which was not clear.

Autopsy: The brain was removed, and upon its removal about $\frac{1}{2}$ ounce of foul-smelling pus escaped through the right frontal lobe. Further exploration showed a double frontal abscess, undoubtedly primary on the right side and extending through the corpus callosum into the left frontal lobe. No other abnormality was found. There was no pus in the ventricles; no internal hydrocephalus.

The pus found in these abscesses contained the *Bacillus pyocyaneus*. The infection from this organism, Dr. Casamajor said, was, according to Oppenheim, most often hematogenous and was one of the commonest causes of brain abscess. The meninges in this case, as well as the frontal and other sinuses, showed no evidence of involvement.

PAVOR NOCTURNUS. TWO CASES. THERAPEUTIC CONSIDERATIONS

By A. Stern, M.D.

The first case was a girl, eight years old, the youngest of seven children. The mother of the child was markedly neurotic; otherwise the family history was negative. This child had always been her mother's pet, rarely leaving her side and without any desire to play with other children. She cried bitterly when her mother went anywhere without her. She had always been fed, dressed and bathed by the mother, who had noticed that while bathing the child, when the mother's hand came in contact with its genitals, the child would press the hand between its thighs. This, Dr. Stern said, we might consider a sexual trauma, as surely we could interpret the child's action in no other light than a desire to prolong the contact, and in this way a desire of a distinctly sexual nature was directed by the child toward the mother. Naturally, the child desired to be whenever it could in the presence of her mother—its loved object, and this was easily enough accomplished by day, while night terrors, from which the child frequently suffered, served the purpose at night.

Night terrors were very properly so called, the speaker said, not only because the children, during the attacks, have such an expression of dread and anxiety on their countenance, but also because these attacks produced terror in the members of the family, and it was through the latter that the child gained its object, namely, to have some one with it at night.

In this case, as in the second case reported, the child was cured of its night terrors by instructing the mother to insist upon its sleeping

alone and paying no attention to it when the attacks occurred. The children were also taught to rely on themselves, to become less self-indulgent and to spend more time in the company of other children.

Dr. Stern said that the practical application of the knowledge gained from Freud's teachings perhaps nowhere else showed such rapid and brilliant results as in the treatment of the different manifestations of the neuroses as they occurred in children. Stekel held the view that the most common form of neurosis that existed in children was the anxiety neurosis, and a very frequent manifestation of this neurosis, and one heretofore very difficult to treat, was the night terror.

The causes of *pavor nocturnus*, as heretofore given, were about as follows: 1. Adenoids and enlarged tonsils. 2. Large and irregular or indigestible meals eaten before going to bed. 3. Terrifying stories told to children before retiring.

In answer to a question as to how the treatment in these two cases differed from that employed heretofore, Dr. Stern said that Holt, in his book, recommended that in cases of night terror where children were very much frightened, some one should be in the room with them, which was directly opposed to the treatment employed in these cases. Drugs had also been recommended, among them bromides.

A CASE OF VAGOTONIA

By Walter Timme, M.D.

Dr. Walter Timme gave a brief survey of the autonomic nervous system, in which he mentioned some of the characteristics of an over-active extended vagus, which were manifested in his patient, such as hyperacidity, hypermotility of the stomach, vomiting, constipation with occasional diarrhea, bradycardia, low blood pressure (105-110 mm.), dermatographism, absent gag reflex, clammy hands and feet and cessation of menstruation. In striking contrast to these symptoms were those of a marked sympatheticotonia, namely, mydriasis and wide palpebral fissures.

The patient was an unmarried woman, 29 years old, who was engaged at social service work. Of some importance in her family history was the fact that two members of her mother's family had suffered from attacks of spasmodic asthma for twenty years. This patient, following a secession of mental shocks in the spring of 1911, began to vomit after each meal, and this daily vomiting had persisted without any lapse up to the present time, a period of over three years. Eight years ago she had a similar attack which lasted three weeks and ceased spontaneously.

The excessive motility of her stomach musculature was proven by two roentgenograms, showing that the stomach emptied itself of bismuth paste in twenty minutes. One of these plates showed very well the condition of the spastic stomach wall termed by Holzkecht "cowhorn" stomach. It was found that the patient could retain some articles of food, notably chocolate, for a much longer period; perhaps this was due to the slightly sympathetic stimulant theobromine which chocolate contained. As the patient was not willing to give up her work, the treatment with atropine could not be carried out very satisfactorily; so much so, in fact, that the small doses of the drug that had been given seemed to bring out a greater opposition on the part of the vagus, increasing some of her symptoms. Various combinations of glandular extracts had been tried with very little

effect. The patient had also been under psychoanalytic treatment without any success.

In reply to a question, Dr. Timme said the patient could retain coffee, if it was strong. As to whether an intra-thoracic thyroid could be ruled out in this case, the speaker said it could not. Vagotonia might be a disease entity, or it might accompany any of the toxic conditions.

Dr. S. Rothenberg, of Brooklyn, said that in the article by Eppinger and Hess on this subject, the authors suggest that it was sometimes possible to bring out an entire train of symptoms by the injection of a small dose of pilocarpin, and the aggravation of certain symptoms thus elicited might act as a guide in the subsequent therapy of the case.

Dr. D. M. Kaplan referred to the work of Fleischmann in the Hess clinic, who came to the conclusion that it was absolutely impossible to classify these cases, owing to the fact that one group of symptoms corresponded to the vagotonic condition while another corresponded to the sympathetic. For example, the dilated pupil had been explained by Eppinger and Hess as due to the presence of excessive adrenal secretion in the blood, and he advised certain tests for ascertaining the amount of the adrenal secretion.

Dr. Timme said the pilocarpin test mentioned by Dr. Rothenberg was only useful in cases where the irritability of the vagus was in doubt. When the vagotonic symptoms were present, the pilocarpin test was unnecessary. As a matter of fact, pilocarpin had been tried in this case and had aggravated the vagotonic symptoms. The speaker said he had seen no instance in which all the symptoms were either wholly vagotonic or sympathetic, and he knew of no one else who had reported a pure case of vagotonia.

Dr. I. Abrahamson thought the word vagotonia was a misnomer in these cases, as the vagus was a very complex bundle of nerves made up of mixed fibers, including both accelerator and depressor fibers to all the viscera. It would be preferable, he thought, to speak of these symptoms as being derangements of either the autonomic or sympathetic systems and to drop the word vagotonia altogether.

PAIN AND TENDERNESS IN SPINAL DISEASE AND ITS SURGICAL TREATMENT

By Charles A. Elsberg, M.D.

Dr. Elsberg showed cases and lantern slides to illustrate the peculiarities in sensory disturbances observed on patients in the course of 100 spinal operations. He emphasized the fact that there was a marked difference between the cervical and the dorsal spinal roots in their structure as well as their course: the cervical roots originated from the cord over an area one to two centimeters wide and ran as separate bundles to the dura, while in the dorsal region the bundles soon united to form one nerve root. Therefore, in the cervical region, a tumor could press on a few of the root bundles and give rise to symptoms from those bundles only, so that one should differentiate between root bundle symptoms and root symptoms. He reported several cases in which pressure upon the lower root bundles of the seventh cervical posterior root produced pain limited to the index finger, so that it was probable that the lower root bundles of the seventh cervical nerve supplied the index finger and perhaps part of the middle finger.

Dr. Elsberg then described the peculiar course of the nerve roots. In the dorsal region the roots first ran downwards, bending upwards again at a very acute angle. Further investigation had shown that the inner surface of the dura of the spinal cord was very sensitive, while the cord itself was insensitive excepting near the origin of the posterior roots, and the posterior roots themselves were very sensitive. Taking these facts into account, it was easy to understand how a small focus of malignant disease in the posterior and lateral surface of the body of a vertebra could cause very acute root pains. Extra-medullary tumors which did not press upon nerve roots might cause pain in the back from pressure on the inner surface of the dura, especially if they laid in front of the slip of the dentate ligament.

The reader then described a peculiar set of symptoms which he had observed in several cases of extra-dural tumor. There was a kind of reverse Brown-Séquard, the motor symptoms being on the opposite side and the sensory symptoms on the same side as the tumor. In these cases there was a collection of fluid under the dura on the side of the tumor, which pressed the spinal cord to the opposite side against the walls of the spinal canal, so that the motor symptoms were due to the pressure of the contra-lateral pyramidal tract against the bony wall of the canal. This could be called *contre coup*, and was similar to what occurred in the posterior cranial fossa when a cerebellar tumor of one side pushed the cerebellum to the other side and caused the facial nerve of the contra-lateral side to be pressed against the petrous portion of the temporal bone, with resulting facial paralysis on the side opposite to the lesion. Finally, he reported peculiar anomalies of the spinal vessels which gave rise to irregular symptoms and which had not hitherto been described. These cases, he said, deserved further study.

In order to illustrate the facts of the paper, Dr. Elsberg presented the following cases:

1. A patient who was completely relieved of symptoms of abdominal pain on the right side by the removal of an extra-medullary spinal tumor from the 12th dorsal segment.
2. A patient from whom a tumor had been removed at the 8th dorsal segment, underneath the slip of the dentate ligament. This tumor had caused paraplegia and severe pain in the back.
3. A patient cured after removal of a tumor from the 8th cervical segment. She had suffered from marked motor symptoms for a long time but sensory symptoms only appeared after lumbar puncture had been done.
4. A patient who rapidly recovered from a paraplegia of three years' duration after the removal of a small tumor from the 6th cervical segment. This patient at first presented symptoms at the 11th dorsal, but a laminectomy failed to discover the tumor. A year later, the patient presented level symptoms at the 1st to 2d dorsal, but the tumor was found at the 6th cervical.
5. A patient who had had spastic paraplegia due to a localized pachymeningitis, with tremendous thickening of the dura at the 6th dorsal level who was completely relieved by excision of the thickened dura.
6. A patient completely relieved for a number of years by decompressive laminectomy from symptoms simulating multiple sclerosis.
7. A patient with acromegaly who had had an acute exudate in the sella turcica, causing the rapid development of eye symptoms. When the patient was admitted to the hospital he had meningeal signs, was blind in

the left eye and had temporal hemianopsia in the right eye. With the subsidence of the hypophysitis or perihypophysitis, the fields of vision in both eyes gradually returned to normal.

Dr. I. Strauss, discussing the last case shown by Dr. Elsberg, said that when he saw this patient, together with Dr. Sachs, the man was in a condition of stupor. He had previously been examined by Dr. Abrahamson, who was then able to make out a hemianopsia in one eye, but when Dr. Strauss saw him the patient was not sufficiently conscious to answer questions. He had then been in the service of Dr. Morris Manges for about a week with a temperature curve very suggestive of typhoid fever, and an examination of the blood showed the presence of the paratyphoid bacillus. He had been suffering for some time from a profuse and offensive nasal discharge. There was increasing stupor and coma, with a slight Kernig's sign and some stiffness of the neck. Repeated lumbar puncture gave clear fluid and no increased leucocytosis: hence it seemed certain that there was no meningeal process. There was a history of trauma eleven years previously (falling from a considerable height) followed shortly afterwards by enlargement of the hands and changes in the face characteristic of acromegaly. The acromegalic features of the case were regarded as due possibly to a traumatic cyst or some other disturbances of the hypophysis. The acute symptoms were regarded as being due to ethmoidal or sphenoidal disease, possibly of paratyphoid origin, though this was not proven. Dr. Emil Mayer was then asked to see the case, and he felt somewhat loath about exposing the sinuses on account of the general blood infections but he was finally convinced that there was no reason why a local focus of infection of this kind, provided it existed, should not be treated just as is done in bacteriemia due to sinus thrombosis in otitic disease. He thereupon opened the ethmoidal cells and drained them. From that time on the patient's improvement set in, and on the very next day his mental condition was much better. It was possible to rouse him, he answered questions intelligently and his temperature gradually reached normal by lysis. At the same time the remarkable improvement in his sight which Dr. Elsberg has shown by his charts, developed.

This case, Dr. Strauss said, seemed to him to present an infection of the sinuses with secondary involvement of the chiasm. The rhinologist not infrequently saw cases of sinus disease, ethmoidal or sphenoidal, with optic neuritis which may go on to blindness, if unrelieved, and the emptying of the sinuses was always followed by a rapid return of vision and lessening of the inflammation of the optic nerve. The speaker said he did not see how the symptoms in this case and their rapid disappearance after operation could be interpreted in any other way. One can be almost certain that there was no meningeal involvement, as proven by the fact that the spinal fluid was clear and that recovery was so speedy.

Dr. L. Hauswirth (by invitation,) who had seen the case in the service of Dr. Morris Manges at Mt. Sinai Hospital, said the sequence of symptoms in this case was rather remarkable, and they finally led to the conclusion that they had to deal with a sinus infection in a patient with acromegaly. The man gave the history of having fallen from a 4-story building 10 years ago and that he grew tremendously after that. When he was admitted to the hospital he stated that for the preceding ten days he had noticed that the sight of his left eye was becoming dimmer and was gradually lost. He then became unconscious, although when Dr. Abrahamson saw him after withdrawing 30 c.c. of spinal fluid he was able to demonstrate a

temporal hemianopsia on the right side and complete blindness on the left. The question naturally arose whether the fall might not have resulted in a hemorrhage, with the formation of a cyst. A peculiar odor was noticed about his bed, and a very offensive discharge was found from both nostrils, particularly from the right. Dr. Emil Mayer's operation on the ethmoidal cells was followed by an immediate improvement in the sight of the left eye; and within twenty-four hours he had a bitemporal hemianopsia which soon disappeared. The failure of the hemianopsia to persist proved either that he had had no cyst at all, or that it had disappeared, an unlikely alternative.

That the infection of the sinus in this case was of paratyphoidal origin, Dr. Hauswirth said, had not yet been proven.

Dr. J. Ramsay Hunt said it was certainly a great pleasure to listen to a surgeon who had had the opportunity of seeing these lesions in the living subject, and who was able at the same time to discuss them from the viewpoint of the neurologist.

Among the lesions described by Dr. Elsberg was an enlargement and varicosity of the veins of the pia-mater, which corresponded in level to the distribution of the symptomatology. In such a condition, the natural presumption would be a secondary congestion of the venous circulation from pathological changes within the spinal cord itself. In this connection it is of interest to recall the rare occurrence of varicose veins of the sciatic nerve.

Speaking of the vertebra pain associated with spinal tumors. Dr. Hunt said he had always explained this on the basis of a localized meningeal irritation. This pain, in his experience, was usually bilateral, and had not the unilateral distribution of the root pains. He thought it well that Dr. Elsberg had emphasized the importance of cutting the posterior root where it had been subjected to pressure for a considerable length of time, and in that connection he asked his advice as to the advisability of cutting these roots for the relief of a long-standing unilateral spasticity, when the site of operation exposed the necessary root distribution. The possibility of contralateral pressure symptoms occurring in connection with lesions of the cord, as shown by the reader of the paper, was of extreme interest. The mechanism of contralateral pressure was recognized as a rare occurrence in the hemisphere of the brain and also in the brain stem, in which event its recognition would be of great importance from the operative standpoint.

Dr. I. Abrahamson said he had seen two cases where marked changes of the vessels of the cord were present. The specimens had not yet been carefully examined in order to determine whether they formed the beginning of an angiomatic condition or a true varix. One specimen did not show any dilatation of the vein—only a moderate hyaline degeneration of its walls. These cases were quite typical in their symptoms, and were in sharp contrast to those common to extramedullary tumor of the cord. In the latter, the motor symptoms were markedly predominant, with root symptoms at the level, while the cases with varicose vessels showed a marked preponderance of the sensory symptoms, the motor symptoms never equalling the sensory in their intensity. The finding of this dilated blood vessel at the exact level and position that was indicated by the signs and symptoms left but very little doubt as to its etiological importance. When one had to deal with a rather atypical level cord picture suggesting an intra-medullary lesion and yet not characteristically so, associated with

slight motor symptoms and marked sensory symptoms, one should be on the lookout for this condition of varix or a type of angioma of the cord.

Dr. Foster Kennedy said that *apropos* of angioma of the cord producing symptoms referable to the interior of the cord rather than to pressure upon the outside of the cord—that is, intra- rather than extra-medullary—he thought it well to bear in mind that a chronic syphilitic pachymeningitis of long duration very frequently produced such circulatory conditions, usually by nipping of the blood vessels, as to cause a breaking down of the interior of the cord, so that we had to deal with a syphilitic syringomyelia. He asked Dr. Elsberg whether he regarded these symptoms as being due to the direct pressure of the varix or to the circulatory disturbance produced thereby; in other words, whether the symptoms were mechanical or circulatory?

Dr. H. Climenko said he had had the opportunity to observe two of these cases in the service of Dr. Abrahamson. In one of them the symptoms closely simulated multiple sclerosis: the sensory symptoms made their appearance later. In the second case he saw the symptoms were also very puzzling for a long time and as it subsequently was shown, the sensory symptoms did not correspond to the location of the tumor. Moreover, there was no constant level, the symptoms shifting up and down. The motor symptoms in this case were so far advanced that even if a tumor had been found, no such improvement as actually occurred would have been expected.

Dr. S. P. Goodhart said that in one of the cases which he saw, the patient gave an early history of paresthesia affecting the thumb and index finger of one hand. It had since occurred to him, the speaker said, that it was very likely that some of the fasciculi of the cord were affected at that time.

Dr. Alfred S. Taylor said that one must be careful lest we attribute too much importance to the presence of these varices. There was a considerable space between the cord and the dura, and he could recall several cases where he had observed large veins in this location, and where a simple decompression had given perfectly satisfactory relief, the condition probably being one of inflammation in the cord and the enlarged vessels being an expression of such inflammation.

Dr. Elsberg, in closing, replying to Dr. Hunt's question as to the advisability of cutting the posterior nerve roots for the relief of spasticity, said he would only advise such a step in cases where the cord had been subjected to pressure for a long time, perhaps four, five or six years, and had been injured beyond the hope of recovery.

As to the significance of these enlarged vessels, he felt convinced they were not the ordinary congested vessels one found accompanying an inflammatory process. In the condition he had described, the veins were from six to eight times their normal size—larger than the radial artery, and at once impressed the operator as enormous vessels of abnormal size. In 114 spinal operations, he had only observed this peculiar enlargement six times; ordinary slight inflammatory enlargement he had seen many times. His object in showing these cases was to call attention to this peculiar anatomical condition that so far as he knew had not been described elsewhere.

SOME PSYCHOLOGIC STUDIES ON THE NATURE AND
PATHOGENESIS OF EPILEPSY

By L. Pierce Clark, M.D.

Dr. Clark referred to the views whether genuine epilepsy was a degenerative organic disease of the cerebral cortex, or a neurosis in its true sense, a functional disease of the nervous system without demonstrable or constant lesions. Both of these views had their strong points and possessed their special advocates. Suffice it to say that probably even less than one half of all cases of genuine epilepsy had a generally acknowledged gross or microscopic pathology or constant lesion. On the other hand, the chemotoxic theory of the disease had even less positive evidence to offer. Even the experimental use of these toxic agents did not produce constant convulsive symptoms. Epilepsy, as we knew it clinically, could not be experimentally induced. There was always something lacking.

In view of the present unsatisfactory anatomic pathology of the disease and an equal failure in chemotoxic pathogenesis, it behooved us to study the clinical makeup of epilepsy more closely. With this view in mind, Dr. Clark said he undertook two years ago to analyze the epileptic temperament and character before the convulsions had occurred. Obviously, cases for such a study had to be selected from those individuals who were already epileptic but in whom it was possible to study the developmental traits of character before the convulsive part of the disorder had developed to complicate the personality makeup. Some 25 cases had now been analyzed carefully by his method. First of all, cases of feeble-mindedness were absolutely excluded, and the majority of those selected were often above their grades at school. The following was a summary of his findings:

It was found that the potential epileptic, as a rule, possessed a supernormal output of energy which was constant and fairly productive of good development results as far as the organic makeup was concerned. There was a poorly repressed or thinly inhibited outcropping of the egotistic tendencies. These characteristics of a pathologic self-love were far beyond the bounds of a pure physiologic variation. As a rule, the potential epileptic was illy adapted to his environment and could not easily change the same. He cooperated badly in social and economic settings. For the most part he was frank; the subtleties of mind were not his; on the contrary, he had a simple childlike pattern of emotional life. He never had the scruples nor doubts of the obsessive neurotic; not being hindered with the inhibitions and prohibitions of the latter class, he was often permitted to become engrossed with the lower animal instincts and passions. With the inhibitions reduced and with an over-emphasized estimate of his own importance and ability, the potential epileptic found himself in constant conflict with the outside world. While the normal individual had his infantile struggle with reality and made the life compromise, the potential epileptic youth kept up the baffling struggle; hence his deep-rooted dislike and even hate of the outside world. Individuals who possessed the epileptic constitution lacked real general interests. The libido was rigid, self-centered and crude. There were small religious promptings; the meaning and ends of life rarely engrossed the potential epileptic. His friendships were perfunctory: the egotistic traits prevented a free range of emotional expression.

A parental attachment of the potential epileptic to his own sex was the

rule and this was especially marked in girls. The love fixation to the mother in the majority was prominent more or less throughout life. The potential epileptic's attitude towards the opposite sex was very significant: it rarely possessed the higher love attributes and more frequently evolved little beyond the simplest or grossest sexual demands. That fact alone, independent of the superadded convulsive disorder, made marriage among epileptics almost invariably a failure.

For some time now it had been customary for psychiatrists to ask themselves, what do psychotic individuals get out of their disease? What purpose do the mental symptoms and settings serve to the unconscious and instinctive demands of the person so afflicted? Such studies had compelled the investigators to discard much of the conscious formulation of the obvious content of the psychosis, and look more deeply into the unconscious strivings as shown in the symbolic utterance and behavior of their patients. With this altered approach to mental disorders a new era dawned in psychiatry; mental symptoms once discarded as meaningless or unimportant took on a new significance and interpretation. From the inspiration of such work, Dr. Clark said, he undertook to study anew the meaning of the fit in epilepsy, a work now some two years old. He now had the material of a dozen or more cases of genuine epilepsy, an epitome of the findings of which he briefly set forth. Uniformly in all cases it was found that the fit served as an unconscious gratification to the libido. By dream analysis, the analysis of many minor attacks and a few major fits, this unconscious striving was often a frank sexual outlet; in others it was a discharge of accumulated pleasure affects that had been long accumulating in the individual epileptic.

The essential motive in all genuine epileptic fits would seem to be at bottom an unconscious striving on the part of the epileptic to return to the intrauterine life in the mother in a state of perfect peace and allmacht, with its insistent urge in the fits, and is conditioned upon the stage of libido fixation in infancy, which occasionally may be so advanced as to reach into homosexuality but rarely into heterosexual life.

Finally, we have more or less definitely indicated what the motive of the unconscious mechanism of the fit really is, but how may we account for the nature and character of the classic but peculiar type of muscular spasm in the grand mal seizure? At another time, Dr. Clark said, he hoped to give a detailed comparative study to show that the types of muscular movements were from that infantile period to which the unconscious urge strives to force the unconsciousness to return, namely, the intra-uterine period and possibly the first few months of extra-uterine existence. The seizure movements were point for point identical with the impulsive movements of the fetus and the period of earliest infancy. In the normally developing child, these muscular movements were slowly but definitely repressed as the will and the conscious life unfolded: in the epileptic the impulsive movement of earliest infancy were released from conscious and voluntary control, and they re-enacted the fetal and infantile purposes of satisfaction of that period.

Lastly, the speaker referred to the enormous therapeutic gain we might obtain in looking at the epileptic mechanism in the light of the therapy briefly set forth in his paper. The hygienic training treatment, the educational and environmental therapy were given a new and clearer meaning by this hypothesis: it signally made clear the reasons for the large majority of epileptics who could not be cured, and, at the same time, it

showed a much more definite and speedier plan by which we might rescue the less severe cases of epilepsy by restoring them to health as shown in an arrest or cure of epileptic fits.

Dr. August Hoch was much interested in Dr. Clark's paper, which was a very valuable contribution, not only to the study of epilepsy, but to the subject of analysis of abnormal mental states in general. On account of the lateness of the hour he desired in his discussion, to confine himself to a few remarks on the general principle of such studies, which, when reported briefly as Dr. Clark did, and outside of the setting of other kindred studies of neuroses and psychoses, often fail to be understood, or, at any rate, do not carry conviction. The most prominent feature which Dr. Clark's study brought out, is that in epilepsy, as is the case in the psychoses and neuroses, that which we might call infantile motives play an important rôle. Such infantile motives are the result of imperfect development of the instincts. Such motives Freud had demonstrated in the neuroses by means of his psychoanalytic method. In psychoses they are very prominent, and, what is more important, quite plain. They express themselves often in delusions and hallucinations of the patient, which we did not understand formerly because the patient does not produce them in an adult logical form, and because we had no idea of the fact that a effective instinct development can give rise to such motives. But once this is known, many of the utterances of the insane become strikingly clear, and do not require the complicated, difficult, and, in the hands of the inexperienced, doubtful method of interpretation, because the patients very plainly express these infantile desires. The interesting personality studies on epileptics which Dr. Clark made, also gave us as all such studies do, an interesting confirmation of the claim that the epileptic presents imperfectly developed instincts. But now we must agree that it is difficult to admit the claim that in patients with constitutional disorder there is an all engrossing attachment to the parents into which even sexual elements enter; but the matter becomes very convincing when we find that this motive returns, with almost tiresome frequency, in the delusions and hallucinations of the psychoses. Still more absurd seems, at first glance the desire to get back into the mother's body, but when looked at from the point of view of a desire for complete shelter, for that most fundamental renunciation of adaptation to reality, it appears much more reasonable, and the special formulation which we find in delusions and in dreams, namely, a formulation which often refers to the real womb, is likely to be, in large part, a secondary mental elaboration. At any rate, this motive has to be accepted as a definite fact, no matter how we try to explain it.

This entire method of studying diseases has added much to our knowledge of the symptom analysis, the structure of the clinical factors, and the forces at work. It should, of course, not be claimed that the forces thus discovered, and the imperfect instinct development, is the final cause of epilepsy. Such studies give no account, and do not claim to give any account, of the organic causes of the constitutional defect, or the organic side of abnormal functioning of the instincts.

Dr. John T. MacCurdy said he had been interested in the problem of epilepsy for some years, and thought that all those who had studied epilepsy with psychoanalytic methods would admit that Dr. Clark's paper marked the most important advance in our knowledge of the subject. We knew from our data, pathologic, clinical and hereditary, that epilepsy occupied a lower scale in mental adaption than did dementia præcox. In

the latter, the essential feature was the attachment of the patient to the parent of the opposite sex. This already implies a certain degree of interest outside the individual; a certain degree of objection. If epilepsy represented a lower stage in adaptation, then we should look for the point of fixation in the stage of "mother's life" and that Dr. Clark had apparently discovered. He tells us that epileptics are intensely egotistical, which points to a failure of objection; that the conflicts of the world, which is another evidence of egoism, were nothing to them; and finally, he has made the very interesting observation that there is a definite desire to return to the mother's body. That is an extraordinary claim, yet we see it in other psychoses. The speaker said he recently saw an epileptic who claimed that during his convulsion he went back to his mother's womb, which he very much enjoyed.

Dr. Smith Ely Jelliffe said that the *mutterleib's phantasy* was met with in various phenomena in addition to the epilepsies such as in various conversation and compulsive phenomena. Dr. Clark apparently would apply a general situation present in the psyche of all individuals as explanatory of a specific phenomenon. It would be of practical interest in the analysis of the epileptic phenomena to see just how the individual had evolved the universally present Oedipus situation through the individual muscle erotic gratification. It was no solution to say that these patients showed an introversion type in phantasy everybody does that. The answer should show how did the epileptics work out their phantasy, not in harmonious muscular activity, but in the discordant and nonadaptive epileptic discharge.

Dr. Clark, in closing, said he had not included the definitely organic cases of epilepsy in his study, and that for the time being he wished to limit his interpretation of the mechanism of epilepsy to that of the so-called idiopathic group. He might say, however, that he had seen several cases of epilepsy which followed in the wake of infantile cerebral palsy in which the mechanism of the attack was quite identical with those he had studied in essential epilepsy as embraced in his paper. He cited the fact that Binswanger, Gowers and Oppenheim had all indicated that the essential makeup in personality and the enduring mental stigma of the disease were, to all intents and purposes, identical in epilepsy following cerebral palsies as compared with that of idiopathic epilepsy. From this and other data in hand he was willing to admit that the possible real genesis of the epileptic state might be organic at bottom. But where persistent attacks occurred, his contention was that the organic insult merely permitted or allowed the mechanism of epilepsy to occur as he tried to show it to have done in his study.

Translations

VAGOTONIA

A CLINICAL STUDY

BY PRIVATDOZENT DR. HANS EPPINGER AND DR. LEO HESS

OF VIENNA

TRANSLATED BY WALTER MAX KRAUS, A.B., M.D., AND
SMITH ELY JELLIFFE, M.D., PH.D.

(Continued from page 176)

It is to be expected that there should exist some hormone in the body which would be an antagonist to adrenalin, and in the light of this we must feel that there is an autonomin which has a selective action upon the autonomic system, just as adrenalin has upon the sympathetic.

However, we know neither its source nor its constitution. But a few substances are known which have a stimulating action upon some parts of the autonomic system. Thyroid stimulates the intestine, the sweat and salivary glands. Infundibulin seems to act only upon the pelvic nerve, leaving other branches unaffected. Formerly it was felt that in cholin, a substance present in every cellular organ of the body, there had been found the universal autonomin. When one considers, however, what large amounts of this substance are necessary to bring about stimulation of the autonomic, it, in contrast to adrenalin, seems very little likely to be able to play a prominent physiological part. Finally we must not omit to remark that substances may be isolated from various organs, which as far as their action upon blood-pressure goes, may be considered autonomic stimulants. The experiments with these substances seem to us not to be entirely reliable, since the substances were never obtained in the pure states. This leads one to believe that the action upon the autonomic system may have been due to the presence of peptone as an impurity. While physiology has yielded us no certain knowledge of any hormone except adrenalin, yet certain disease complexes give us a hint

where autonomic stimulants are produced. It has been felt that extirpation experiments will show the way by which to approach the sought-for goal.

Up to now this method has only been of value in metabolic questions. On the basis of this method, it has been ascertained that the pancreas may be the site of production of autonomic stimulation. Its secretion probably acts in opposition to that of the chromaffin system, since when it is absent, the full action of the chromaffin hormone is felt, at least, as far as metabolism is concerned. Depancreatized dogs showed neither tachycardia nor increased blood-pressure. Only as far as the pupil is concerned are we certain of any increase in the tone of the sympathetic system. The thyroid must, as far as metabolism is concerned, furnish sympathicotrophic substance, since feeding thyroid causes an increase in metabolism as does adrenalinemia, and also favors alimentary glycosuria, *i. e.*, lowers sugar tolerance.

Lack of thyroid, on the other hand, increases tolerance for carbohydrates, and furthermore this tolerance may remain the same in spite of administration of adrenalin.

We could present many other facts which go to prove that the various organs produce sympathicotrophic and vagotonic impulses, but only those facts relative to metabolism can be presented, while the action upon the tone of smooth musculature and upon the activity of the other glands can scarcely be more than touched upon. We may expect by analogy to the action which we know is exerted upon metabolism by the glands of internal secretion, that they would also exert an inhibitory or stimulative action upon other vegetative functions.

The question now arises whether some clue may not be derived from general pathology, which may strengthen our idea that in the absence of certain endocrinous glands, there exists some condition analogous to vagotonia. The best example of this is Addison's disease, in which the organ from which sympathicotrophic impulses arise, is absent. If we test the symptoms of this disease, we find the following: emaciation, diarrhea, lowered blood-pressure, pigmentation, sweating, general adynamia, high sugar tolerance and frequently hypoglycemia.

The diminution in blood-pressure and blood sugar is surely referable to the lessened activity of the chromaffin system. In addition there are conditions like those resultant upon autonomic

stimulation: Sweating, diarrhea, which in comparison to their insignificance in the basic disease, tuberculosis, are very prominent. There are anatomical findings in typical Addison's disease besides those in the adrenals, *i. e.*, a general lymphatic enlargement. The thymus also is enlarged, it being not only persistent in adults, but also not infrequently hyperplastic. The question of whether or not the thymus belongs to the lymphatic system will not be discussed in this paper. This fact must be added, that while the clinical picture of Addison's disease always is associated with diseased adrenals, diseased adrenals may not always cause a symptom-complex identical to Addison's disease. One is not infrequently astonished to find at autopsy that the adrenals are severely diseased, a diagnosis which could not be made *in vivo* due to the lack of any significant symptoms. And furthermore, every case does not show, at section, an enlarged lymphatic system and a hyperplastic thymus. Finally, adrenalin injections benefit the symptoms of Addison's disease.

This coincidence between adrenal disease and lymphatism or thymus hyperplasia was first observed in the cadaver by Wiesel,³² Hedinger³³ and Hart.³⁴

These authors suggest that apparently primary lymphatism might be due to inferiority of the chromaffin system. Wiesel stated that the adrenalin-producing system was poorly developed just in that group of cases in which status thymicus was most marked. The question now arises, what knowledge concerning this relationship may be gained by considering the clinical and anatomic findings in Addison's disease. If the conception of vagotonia as the opposite to that constitutional state in which the adrenalin-producing organs dominate metabolism and smooth muscle activity be correct, there can be no doubt that Addison's disease may be considered as an exaggerated form of vagotonia.

Unfortunately our knowledge of the functions of the autonomically innervated organs is as yet very scanty.

This we have been able to augment a little. Besides we must not overlook the probable effects of a state of inanition, such as found in Addison's particularly upon the functions which the

³² Wiesel, J., *Virchow's Archiv.*, 176, p. 103, 1904; *Beiträge zur pathol. Anatomie und allg. Pathologie*, 37, p. 168, 1904.

³³ Hedinger, *Verhandl. der XI Tagung der Deutschen pathol. Gesellschaft*, p. 29, 1907. *Frankfurter, Zeitsch. f. Pathologie*, 1, p. 527, 1907.

³⁴ Hart, *Weiner klin. Wochenscht.*, 21, p. 1119, 1908. *Zentralblatt für die Grenzgebiete der Medizin und Chirurgie*, 12, p. 321, 1909.

smooth muscles subserve. Thus we never have a truly clear-cut picture. But in spite of this the symptoms of vagotonia are so outspoken in Addison's disease that we must of necessity regard it as a general secondary vagotonia. In spite of the inanition, hyperacidity and evidence of increased gastric tonus are not infrequently found in Addison's. The enormous development of the lymphatic tissue and the frequency of thymus hyperplasia, found in autopsy, in those who have died of Addison's, and similar findings in those who clinically were vagotonics, lead us to believe that a relationship exists and that perhaps the manifestations of vagotonia are related to the pathological conditions in the thymus and the marked enlargement of the lymphatic tissues.

(To be continued).

Pertiscope

Archiv für Psychiatrie und Nervenkrankheiten

(51 Band, 3 Heft)

- XXIV. Cases of Optic Nerve Atrophy in Tabes and General Paralysis. STARGARDT.
XXV. A Study of the Pupils in the Mentally Ill and in the Normal. RUNGE.
XXVI. The Significance of Gynecological Diseases and the Effect of their Cure on the Psychoses. KÖNIG AND LINZENMEIER.
XXVII. The Value of Psychoanalysis. A. HOCHÉ.
XXVIII. The So-called Outer Glandular Layer in Acquired Cerebellar Disease. L. BÉRIEL.
XXIX. The Combination of Muscular Dystrophy with Other Muscular Diseases. OTTO KLIENEGER.
XXX. The Relationship of Psychiatry to the Other Special Fields of Medicine. W. H. BECKER.

XXIV. *Optic Atrophy in Tabes.*—Stargardt writes a monographic article on the disputed question of optic atrophy in tabes and progressive paralysis. He concludes that the changes do not take place primarily in the retina, as has hitherto been assumed, but that in the diseases under consideration the cause must be sought in some part of the optic tract. He also reaches the definite conclusion, important for therapeutic reasons, that optic atrophy should no longer be regarded as due to a metasymphilitic poison, but rather as a direct action of the spirochetes. The conclusion is inevitable, that every possible means should be taken to combat the disease at its source, through salvarsan combined with mercury. The difficulty is through chemical means to reach the actual foci in the nerve tissue. Whether or not a more direct method may be practiced is still open to question.

XXV. *Pupillary Reactions.*—Runge attempts a study of pupillary phenomena as observed in mental disease and in healthy individuals. Following the more recent work of Bumke, the writer has investigated the finer movements of the iris in a great variety of conditions, both normal and pathological. The details of this investigation are not possible to give in the scope of a brief review on account of the large amount of statistical material used. In general, it appears that certain definite conclusions may be reached regarding the pupils under various conditions of disease apart from the well-recognized Argyll-Robertson type. The physiology of the reflex is discussed at length.

XXVI. *Gynecology and Psychoses.*—König and Linzenmeier reach the following general conclusions from a study of gynecological conditions in relation to the psychoses: Proof that psychoses are caused by such conditions is not forthcoming; how far ovarian secretion may have relation to mental disturbances is as yet not known; local cure has no influence on the course of a psychosis; the question of suggestion in such cases is important; gynecological examinations and treatment may be

practised on mental cases without danger, but are only indicated for purely local reasons; it is undesirable to attempt to restore menstruation; operative intervention should be undertaken when possible under conditions of complete narcosis.

XXVII. *Psychoanalysis*.—Hoche makes a somewhat bitter attack on the principles of psychoanalysis as enumerated by Freud and his followers. He believes that psychoanalysis is justified neither on theoretical nor empirical grounds; that its therapeutic efficiency is unproved; that its permanent value for clinical psychiatry amounts to nothing; that the impression it has made depends upon thoroughly unscientific methods; that the practice of psychoanalysis as now largely used is a danger to the nervous system of the patient and a reflection upon the medical profession; and finally, that the only permanent interest in the subject lies in the field of its relation to the development of culture in the broad sense.

XXVII. *Glandular Layer in Cerebellar Disease*.—Bériel offers a brief histological study of the outer layer of the cerebellum on the basis of the investigation of a single case suffering from paraplegic spastic disturbance. A special study is made of certain encephalitic areas on the surface of the cerebellum.

XXIX. *Muscular Dystrophy*.—In this article, Klieneberger gives the clinical history of a case of muscular dystrophy with myotonic disturbances combined with painful paresthesia and certain other disturbances. The article is a significant contribution to the obscure subject of the muscular diseases.

XXX. *Psychiatry and Medicine*.—Becker outlines the relations of gynecology, obstetrics, internal medicine, ophthalmology, otology, dermatology, and dentistry, and shows (as it is not difficult to do) the important bearings which each of these special fields in medicine have upon the mental life of the individual.

E. W. TAYLOR (Boston).

Deutsche Zeitschrift für Nervenheilkunde

(51 Band, 1-2 Heft)

1. Some Unusual Manifestations in Hemiplegia. PANSKI.
 2. The Diagnosis and Therapy of Tumors of the Parietal Lobes. VÖLSCH.
 3. The Symptomatology of Cerebral Paralysis. BERGMARK.
 4. A Case of Acute Hemorrhagic Encephalitis of the Pons with Extensive Bleeding. BERG.
 5. A Case of Syphilitic Spinal Amyotrophy of the Shoulder Girdle. KUMMANT.
 6. Recurrent Polyneuritis. HOESTERMANN.
 7. A Further Contribution to Our Knowledge of Muscle-cramp, Peripheral Origin and Relation of Symptoms. BITTORF.
1. *Unusual Manifestations in Hemiplegia*.—The writer without considering the relative frequency of these manifestations, speaks of disturbance of taste, furred tongue and blisters, pseudobulbar palsy and diabetes.
 2. *Tumors of the Parietal Lobes*.—In the matter of diagnosis, emphasis is laid upon the early appearance of spastic hemiparesis and dissociation of sensation. Finally, the new and much spoken against method of brain puncture should be resorted to.
 4. *Hemorrhagic Encephalitis of the Pons*.—Berg tells of a perplexing

case and one of unusual interest where death occurred suddenly in a young and healthy individual. The autopsy report and microscopic findings are detailed. Clinically, there were spastic symptoms in the left arm and leg, right sided flaccid paralysis with irritative symptoms of the right oculomotor nerve. These symptoms pointed to an affection of the pons. The attack was apoplectiform in nature and in the absence of evidence pointing to thrombosis or embolism, the presence of hemorrhage was suspected. This was confirmed at necropsy. During the course of the disease, a lumbar puncture yielded fluid sanguinolent in nature, that is, the blood was uniformly mixed and not coagulated; this also pointed to a "natural" hemorrhage.

5. *Syphilitic Spinal Amyotrophy of the Shoulder Girdle*.—The data upon which this diagnosis was based are: (1) Twenty years before there was a luetic infection. (2) Since the last two years there had developed a progressive atrophy chiefly of the musculature of the shoulder girdle, with the cerebral symptoms of headache and dizziness, with the spinal symptoms of bowel and bladder disturbance, together with exaggerated reflexes and sensory changes. (3) A positive Wassermann in the blood and cerebrospinal fluid. (4) Positive result in Nonne-Apelt reaction and lymphocytosis amounting to 30 in cmm. of cerebrospinal fluid. (5) Slight increase in temperature.

7. *Muscle-Cramps*.—This further contribution speaks of muscle cramps as occurring in the conditions of myokymia and myotonia, in alcoholism, in exposure to intense heat and in muscular rheumatism. The cause is for the most part a toxic substance causing a neuritis or a neuromyositis. YAWGER (Philadelphia).

THE PYRAMIDAL TRACT IN THE RED SQUIRREL (*Sciurus hudsonius* Loquax)
AND CHIPMUNK (*Tamias striatus* Lysteri). Sutherland Simpson.
(The Journal of Comparative Neurology, Vol. 24, No. 2.)

The cortical motor areas in the left cerebral hemisphere were extirpated in three red squirrels (*Sciurus hudsonius* Loquax) and three chipmunks (*Tamias striatus* Lysteri) and the resulting degeneration followed by the Marchi method.

The pyramidal tract occupies the usual position in the crusta, pons and medulla oblongata until the decussation is reached. Here, in the lower part of the bulb, the fibers cross the middle line abruptly in bundles which interlace with those of the sound side and pass through the gray matter into the funiculus cuneatus where they turn caudalwards to enter the spinal cord and form the crossed pyramidal tract. The decussation is complete, no fibers remaining on the same side when the spinal cord is reached. There is no sign of a crossed lateral tract. The dorsal tract can be followed as far as the lower sacral segments. A considerable number of degenerated fibers disappear in the upper cervical segments of the spinal cord.

JELLIFFE.

Notes and News

THE PHILADELPHIA POST-GRADUATE SCHOOL OF NEUROLOGY

The neurological wards of the Philadelphia General Hospital offer unusual facilities for post-graduate instruction in nervous disease, these wards containing about four hundred patients illustrating all forms of organic, functional, and psychopathic disease. The Philadelphia Hospital for the Insane, which is a part of the General Hospital and situated on the same grounds, has more than two thousand patients.

All the members of the neurological staff of the Philadelphia General Hospital are connected with neurological services in other hospitals and institutions, which will enable them to supplement the instruction given at the hospital. With the approval and encouragement of the Director of the Department of Public Health and Charities, Dr. Richard H. Harte, the neurological staff has organized a post-graduate school of neurology. The time is opportune for this step. Owing to the terrible war in Europe, it will probably be one or two years at least before American students can avail themselves of the neurological clinics and laboratories of London, Paris, Vienna, Berlin, Rome, and other centers of medical instruction abroad.

Instruction will be arranged in four periods of six weeks each, one, two, three or four of which can be taken by the students. The first course of six weeks will begin Monday, December 7, 1914, and continue until the end of January. The second and third courses will be in February, March, April, and May. Special short courses of two or three weeks each can be arranged for the months of June, July, August, and September. The instruction will be given by the members of the neurological staff of the Philadelphia General Hospital and their assistants.

The staff is composed of eight members, four of whom are on duty from August 1 to February 1, the other four from February 1 to August 1. A limited number of clinical lectures will be given, but in the main the instruction will be in the nature of ward visits and conferences. Short special courses on serology, pathology, neurological ophthalmology, and the jurisprudence of nervous disease and insanity will be offered in connection with the clinical teaching. The time of the students will be fully occupied.

The fees for each period covering all branches taught will be twenty-five dollars. The only extra fees will be in courses like serology and pathology where an amount sufficient to cover the necessary expenses will be charged.

The faculty is composed of the following members of the neurological staff of the Philadelphia General Hospital: Dr. Charles K. Mills, 1909 Chestnut Street; Dr. Charles W. Burr, 1918 Spruce Street; Dr. William G. Spiller, 2046 Chestnut Street; Dr. James Hendrie Lloyd, 116 South 21st Street; Dr. Charles S. Potts, 2018 Chestnut Street; Dr. D. J. McCarthy, 2025 Walnut Street; Dr. T. H. Weisenburg, 2030 Chestnut Street; Dr. George E. Price, 1700 Walnut Street.

Dr. Francis X. Dercum, 1719 Walnut Street, consulting neurologist

to the hospital, and a former active member of the staff will take part in the instruction.

For information address

DR. CHARLES K. MILLS, *Dean*,
1909 Chestnut Street,
Philadelphia.

The Los Angeles County Psychopathic Hospital, recently erected upon the grounds of the County Hospital, has been opened as a psychiatric department of this institution. It can comfortably accommodate about seventy-five patients and has a complete hydrotherapeutic equipment. It is intended to serve as a place of detention for persons suspected of being insane until their cases are investigated and a disposition is made of them, to furnish treatment for acute cases likely to recover within a short time, to serve as a center for the dissemination of information with regard to insanity among the general public and as station for field work. It will also furnish to students and physicians excellent facilities for the study of psychiatry since the psychiatric material of Los Angeles is unsurpassed anywhere in the west.

The hospital is under the general direction of Superintendent Whitman with Dr. C. L. Allen as psychiatrist in charge.

PROGRAM OF THE AMERICAN NEUROLOGICAL ASSOCIATION

Post Natal Growth of the Brain Under Several Experimental Conditions.

By Drs. Donaldson, Hatai and King.

The Relation of Structure and Function in the Nervous System. By Dr. Stewart Paton.

Ventricular Hemorrhage. By Dr. Alfred Gordon.

Alexia and Astereognosis. By Dr. F. R. Fry.

Report of Two Cases of Epilepsy with Unusual Vascular Findings. By Dr. E. Sachs.

The Paralytic and Other Persistent Sequelæ of Migraine. By Dr. J. Ramsay Hunt.

Report of a Case of Pituitary Tumor. By Dr. W. M. Leszynsky.

Note on the Physiology of the Pars Anterior of the Pituitary Body. By Dr. Alfred Reginald Allen.

Hormone Therapy in So-called Nervous Disease. By Drs. Joseph Collins and Henry Marks.

Abderhalden Reactions and Defective Mental and Physical States. By Drs. S. D. W. Ludlum and Corson White.

Dilatation of Cerebral Ventricles in Various Functional Psychoses. By Dr. E. E. Southard.

The Intelligence Tests in the Psychoneuroses. By Dr. C. L. Dana.

The Study of Temporary Abnormal Mental State, Conforming to Recognized Types of Psychoses. By Dr. S. I. Schwab.

The Development and Operation of the Laws for Hospital Observation of Cases of Alleged Mental Disease or Defect, in Massachusetts. By Dr. Henry R. Stedman.

Mental Disturbances in the Feeble-Minded. By Dr. W. N. Bullard.

The Mechanism of the Tic Neurosis. By Dr. L. Pierce Clark.

Clinical and Kinematographic Observations Illustrative of the Barany Methods in the Study of Vestibular and Cerebellar Disease. By Drs. T. H. Weisenburg and I. H. Jones.

- Some Observations on the Use of the Barany Tests in Vestibular, Cerebellar and Cerebral Disease. By Dr. C. K. Mills.
- Huntington's Chorea: Correlation of Hereditary and Pathological Factors. By Dr. S. E. Jelliffe.
- The Family Form of Pseudo-sclerosis and Other Conditions Attributed to the Lenticular Nucleus. By Dr. W. G. Spiller.
- A Case of Wilson's Disease, Progressive Lenticular Degeneration, with Pathological Findings. By Drs. F. Tilney and G. M. MacKenzie.
- Lenticular Degeneration. A Report of a Case with Autopsy. By Dr. J. H. W. Rhein.
- A Brief Review of a Year's Neuro-surgical Work, with Special Reference to Spinal Cord Lesions. By Drs. B. Sachs and C. A. Elsberg.
- Some Abnormalities of the Posterior Spinal Vessels; Their Symptomatology and Treatment. By Dr. C. A. Elsberg.
- Preliminary Report on the Treatment of Paresis by Injections of Salvarsan and Definite Doses of Neosalvarsan into the Lateral Ventricle. By Drs. G. M. Hammond and Norman Sharp.
- Observations on Hereditary Syphilis Affecting the Nervous System. By Dr. C. D. Camp.
- Diagnosis and Treatment of the Various Forms of Meningitis. By Dr. H. A. Cotton.
- Circumscribed Purulent Meningitis Limited to Frontal Lobe; Due to Sinusitis. By Dr. Samuel Leopold.
- Meningitis Sympathica. By Dr. Israel Strauss.
- Histopathological Findings in a Case of Landry's Paralysis. By Dr. E. D. Fisher.
- A Case of Central and Peripheral Neuro-fibromatosis (von Recklinghausen's Disease). By Dr. Peter Bassoe.
- Spondylitis. By Dr. B. Sachs.
- The Nature and Location of Tumors of the Spinal Cord. By Dr. C. L. Dana.
- A Frequency List of Mental Symptoms Found in 17,000 Institutional Psychopathic Subjects (Danvers State Hospital, Massachusetts). By Dr. E. E. Southard.
- The Development of the Brain from the Stage of Neural Plate to Term as Shown by Borne Reconstruction Models Obtained from the Domestic Cat. By Drs. F. Tilney and H. von W. Schulte.
- Syphilis of the Nervous System without Pathognomonic Signs. Its Frequency of Occurrence and Clinical Display. By Dr. Joseph Collins.
- The Tardy or Late Paralysis of the Ulnar Nerve. By Dr. J. Ramsay Hunt.
- The Supra-optic Canal in its Probable Relations to the Phenomena of Choked Disc. By Dr. F. Tilney.

The Journal OF Nervous and Mental Disease

An American Monthly Journal of Neurology and Psychiatry, Founded in 1874

Original Articles

A STUDY OF THE SPINAL CORD IN A CASE OF ISOLATED ATROPHY OF THE SMALL MUSCLES OF THE HANDS*

By MARY ELIZABETH MORSE, M.D.

The purpose in reporting this case is twofold: first to describe the pathological changes in the cord in a case of isolated atrophy of the intrinsic muscles of the hand, and secondly to locate the cell groups for these muscles in the cord.

Pierre Marie and Foix¹ have recently distinguished among the muscular atrophies a group, characterized by strict limitation to the small muscles of the hand, with unilateral predominance, of slow evolution, and non-progressive nature. The authors have collected 13 such cases, 4 with autopsy. They divide them into two groups according to the presence or absence of signs of syphilis in the nervous system. One half of their cases were syphilitic, the other half probably not so. The lesion found was a localized destruction of the anterior horn, unaccompanied by marked inflammatory or neuroglia reaction, at the level of the lower half of the seventh cervical, the eighth cervical and the upper half of the first dorsal segments. To this condition the authors gave the name "anterior tephromalacia." In the syphilitic cases the meningo-vascular lesions are those usually found

* From the laboratory of the Worcester State Hospital, 1915-1.

in spinal syphilis. The meningeal lesions and perivascular infiltration may however be absent, leaving only a considerable thickening of the walls of the arterioles. The authors therefore conclude that senile arteriosclerosis is here capable of producing lesions analogous to those of syphilis. The white matter is almost entirely normal. The cause of the lesion in all cases, the authors believe, is a progressive ischemia, due to a thickening of the vessels in the territory supplied by the sulco-commissural arteries.

Leri,² in discussing the frequency of syphilis in the etiology of the various muscular atrophies, considers this form to be of luetic origin, and mentions that it may be found in connection with an Argyll-Robertson pupil, a positive Wassermann reaction in the serum, or lymphocytosis in the cerebrospinal fluid. He calls the condition a true vascular syndrome of the anterior horns.

In regard to the second point raised by the present case, i. e., the localization in the cord of cell groups for various muscles, it may be said that the question has been studied experimentally in animals by observation of the location of the degenerated nerve cells following the excision of single muscles or groups of muscles, the section of nerves, or disarticulation at various joints. In man conclusions have been drawn chiefly from amputations. Bikeles³ researches have shown that the cell groups which degenerate in consequence of the resection of a given nerve have essentially the same location in the dog, cat and monkey. The results obtained from animal experiment agree with those found in the human cord, as far as the latter has been studied. In man the topography of the lumbosacral enlargement is better known than that of the cervical, in the first place because more lumbosacral cords have been studied, and in the second because the cell groups are here more detached than in the cervical. On account of its strictly differential character, the present lesion offers an excellent opportunity, and one which has apparently not been utilized, of determining the position of the cell groups for the muscles of the hand.

The intrinsic muscles of the hand are supplied by the ulnar nerve, with the exception of the abductor, the opponens, the outer head of the flexor brevis pollicis, and the two outer lumbricales. The latter group is innervated by the median, which arises from the fifth to the eighth cervical and the first thoracic nerves. The

ulnar is derived from the eighth cervical and first thoracic nerves. As the upper part of the origin of the median doubtless represents the muscles of the forearm, it is particularly in the lower part of the cervical cord and in the first dorsal segment that lesions may be expected in atrophy of muscles of the hand.

The cell groups⁴ that are present in this region, for either the whole or a part of its extent, are the anterior and posterior mesial, the antero-lateral, the postero-lateral, and the post-postero-lateral. The antero-mesial column is small from the fifth to the eighth segment, but in the lower part of the eighth and in the first dorsal attains large size. A posterior mesial group cannot be differentiated in all sections. The antero-lateral group (lower part) reaches its greatest development in the seventh, then gradually diminishes, and disappears at the lower part of the eighth segment. The posterolateral column is at its maximum in the fifth and sixth segments, decreases in the seventh, to increase again in the upper half of the eighth. The post-posterolateral group appears first in the eighth, behind the inner part of the postero-lateral column. It increases in size throughout the lower part of the eighth, coincidently with the diminution of the posterolateral group. It continues through the first dorsal, where it forms the prominent posterolateral angle, and disappears at the second dorsal segment.

There is general agreement that the mesial group is the cell column for the trunk musculature,⁵ the anterior mesial representing the muscles anterior to the vertebral column, e. g., the scalenii, the ileo-psoas, etc., and the posterior mesial group the long muscles of the trunk. The anterolateral group is probably connected with the anterior muscles of the neck, the intercostals and the shoulder and pelvic musculature. The posterolateral columns are the centers for the muscles of the limbs. There is evidence that the post-posterolateral group in the cervical cord is concerned with the muscles of the hand, and its homolog in the sacral cord with those of the foot. Marinesco⁶ found degeneration in this column after disarticulation of the forepaw in a dog, and also in a human case following amputation of the fingers. Van Gehuchten and Nelis⁷ observed chromatolysis of all the cells of this column in the sacral cord of a human case after amputation of the feet, and the former author therefore calls it "the nucleus of innervation of all the intrinsic muscles of the foot."



FIG. 1. Upper part of eighth cervical, showing difference in size and shape of anterior horns and loss of cells in the right posterolateral and post-posterolateral groups.



FIG. 2. Lower part of eighth cervical.

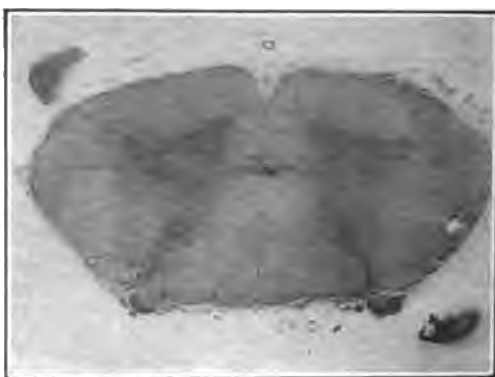


FIG. 3. Upper part of first dorsal. Almost complete devastation in right post-posterolateral group.

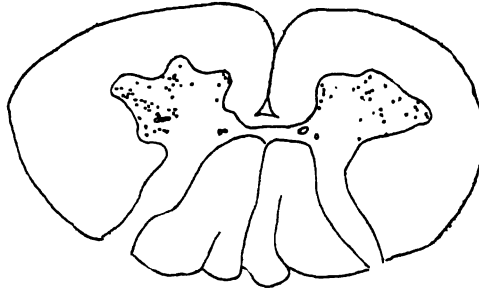


FIG. 1a. Projection drawing of same section.

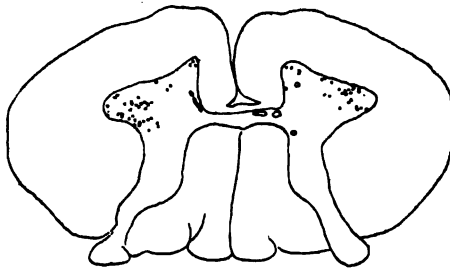


FIG. 2a. Projection drawing of the section following Fig. 2.

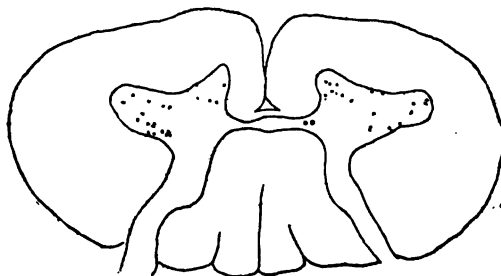


FIG. 3a. Projection drawing of a section near Fig. 3.

The cell groups for the different nerves of the limbs are, however, not sharply defined. According to Marinesco⁸ the nucleus of the ulnar and that of the median are fused into a single mass, which may be explained by the fact that embryologically the ulnar nerve is an offshoot of the median. Marinesco locates this common nucleus, at the level of the seventh segment, at the posterior border of the anterior horn, in a line extending from the postero-external angle to the posterior horn. The cells of the median are placed at a higher level than those of the ulnar, the latter predominating in the eighth cervical and first dorsal. In the eighth cervical the cells of the ulnar extend around onto the external border of the anterior horn, while at the level of the first dorsal they are placed exclusively in the posteroexternal group of the posterolateral column. Bikeles and Franke⁹ after resection of the median and ulnar nerves found the degenerated cells in the median part of the posterolateral group in C7 and C8, where they reached the posterior border of the horn.

CASE: The present case is that of a man, 58 years old, who was admitted to the Worcester State Hospital, June 9, 1913. The past history was not obtained, but it was stated that the patient had used alcohol to excess for 25 years. On admission he was depressed and apprehensive with a sense of impending death; retrospective and self-accusatory. Physical examination revealed nothing noteworthy except the condition of the hands.

Description of the Hands.—The right hand bore a large scar, with considerable fleshy deformity, on the ulnar side of the wrist, and also showed the loss of the fourth and fifth finger tips. There was very marked atrophy of the muscles of the thenar and hypothenar eminences and also some atrophy of the interossei. The left hand showed a slighter atrophy, but a fairly definite shrinkage of the thenar and hypothenar eminences. No evidence of atrophy of the interossei on the left.

The patient died ten days after admission of bronchopneumonia. The psychiatric diagnosis was the depressed phase of manic depressive insanity.

Autopsy, two hours after death, showed in addition to bronchopneumonia, a moderate sclerosis of the aorta with a few calcified plaques. The calvarium was very thick and dense. The dura was slightly tense, opaque, and somewhat thickened. There was a small amount of clear subpial fluid. The brain weighed 1,435 grams and was of a uniformly soft consistence, no focal lesions of any type. The ventricular floors were smooth. The basal vessels showed slight sclerosis.

Microscopic examination of the trunk organs added nothing of importance.

Technical Methods.—Sections from the pre- and post-central, the frontal, temporal, occipital and hippocampal cortices, the cerebellum, the medulla and the upper cervical, middorsal and lumbar regions of the cord were stained by the Nissl and Weigert methods and by Mallory's phosphotungstic acid hematoxylin method for neuroglia. The remainder of the cord was fixed in formalin, and the region from the upper part of the seventh cervical through the second dorsal was embedded in paraffin, cut in serial sections of 6μ thickness, and stained in thionin. Every fifteenth to twentieth section was described, and the cells in the various groups on each side counted.

Examination of the Nissl sections from the various cortical areas gave the following results. The pia in the intergyral spaces shows a moderate increase in fibrous tissue and a sprinkling of lymphocytes and large endothelial cells. Cortical architecture is normal. The nerve cells, with the exception of those surrounded by satellites, stain well, contain a good complement of Nissl bodies and small amounts of lipochrome. Many of the cells in the infrastellate layers are surrounded by satellites. This reaction is most marked in the postcentral and frontal cortices and in the deeper parts of the pyramidal layer of the hippocampus; less marked in the temporal and precentral regions, and does not exceed normal in the occipital cortex. The vessels, both pial and cortical, are considerably thickened. The great majority of them are free from exudate, although a slight excess of lymphocytes may be detected in a few places. In the white matter there are small perivascular collections of green pigment. The sections stained for neuroglia show a moderate general thickening of the glial mat, with some extension into the zonal layer, considerable perivascular gliosis and an increase of glia nuclei in the white matter. The Weigert sections are not remarkable. Sections of the medulla at the level of the upper and the middle parts of the olives, and of the decussation of the pyramids show a moderate thickening of the peripheral glia, normally staining nerve cells, vascular thickening as in the cortical areas, groups of satellites about a few cells and a slight lymphocytic reaction about a few vessels.

Spinal Cord.—Sections from the third, fourth, fifth and sixth cervical segments show no difference in the representation of the cell groups on the two sides. There are, however, scattered atrophic cells on both sides in all segments. The vessels, both in the pia and cord, are considerably thickened, but are free from exudate. There is a peripheral gliosis about the anterior fissure and at the point of entrance of the posterior roots.

Description of the Lesion.—Beginning in the upper part of C7

with serial sections, there is apparent macroscopically in the first third of the segment a slight flattening of the right anterolateral border of the cord. There is no difference in the size of the horns, but the right is more nearly rectangular than the left, due to lack of projection of the anterolateral, and more particularly the posterolateral group. Microscopically a few abnormal cells are found distributed through the anterior horns. These cells are shrunken and angular, have visible processes and stain deeply, but as a rule they contain Nissl bodies. In the central regions of the anterior horns are a number of small nerve cells surrounded by clusters of eight or nine satellites. Counts of cells in the mesial and antero and posterolateral groups show no significant differences between the sides. There is a focus of lymphocytic infiltration about the vessels in the anterior fissure, extending vertically through half a dozen sections, and also a sprinkling of lymphocytes about several small arteries in the anterior horns. Areas of hemorrhage are present in the gray matter. The corpuscles in these are intact and there is no cellular reaction or pigment formation associated with them.

In the middle third of C7 a slight but definite narrowing at the root of the right anterior horn, as compared with the left, becomes perceptible macroscopically. This corresponds microscopically to a cell loss in the posterolateral group, which is evident both on inspection and by cell counts. The diminution of cells is by no means constantly found, the right posterolateral group often showing as many or occasionally even more cells than the left. The gaps are situated sometimes at the tip of the posterolateral angle, sometimes along the posterior border of the horn, or in both locations. Cell counts in the other columns show no important differences on the two sides.

In the lower third of C7 cell losses in the above mentioned locations become more numerous on the right side. There is also in a few sections a suggestion of a scarcity of cells in the posterolateral groups on the left. The anterolateral group on the right appears in a number of sections to be poorly provided with cells in comparison with the left side. Quite marked lymphocytic infiltration is present at one point about a sulcal artery and its branch to the right anterior horn; less marked around that to the left horn.

The lesion increases in C8, but cell counts show that both posterolateral and post-posterolateral are less affected in the middle than in either the upper or lower thirds of the segment. The meaning of this is not clear. The lesion increases suddenly at the lowest portion of C8. Throughout the segment the right anterior horn is smaller than the left in all its dimensions and the right posterior horn is also more slender than the left. Atrophic cells are numerous in all groups, and a number of cells showing

the axonal reaction are present in the anteromedial groups. There are also a few faded blurred cells with finely granular cytoplasm and nuclei which stain faintly or not at all. Small collections of lymphocytes accompanied by phagocytes laden with olive green pigment occur not only about vessels in the anterior horns, but also occasionally in the posterior horns. Hyaloid droplets are found both in the gray and white matter.

The shrinkage of the right anterior horn is due to a loss of cells in a definite situation, which is at the posterior surface of the anterior horn at its junction with the posterior horn, and in a slightly smaller number of sections along the posterior border of the anterior horn. In some sections there is loss in both these regions, and in an occasional section a gap in the group at the tip of the posterolateral angle. On the left side also gaps occur in a number of sections in the same groups. The cell losses, are, however, almost exclusively at the junction of the anterior and posterior horns, and do not extend to the posterolateral angle.

The post-posterolateral group contains in the majority of sections of C8 fewer cells on the right, but on the other hand, as has been said, there are sections in which it shows a poor representation of cells on the left.

The maximum of the lesion is reached in the lower part of C8 and the upper third of D1. The anterior horns show here their greatest difference in size. The anterior border of the eighth is almost straight instead of exhibiting the cup-shape, normal for the lower half of C8. The posterolateral angle is very sharp and the posterior border is flat, instead of showing the usual convexity, due to the post-posterolateral column.

In the lower third of C8 the right posterolateral group gives smaller cell counts than the left in 79 per cent. of the slides examined, in the upper third of D1, in 33 per cent., in the middle third in 20 per cent. The difference between the posterolateral groups decreases, therefore, as the group wanes. The brunt of the damage in the upper third of D1, is borne by the post-posterolateral group, which contains fewer cells on the right in 100 per cent. of the slides examined, as contrasted with 59 per cent. in the lower third of C8, and 67 per cent. each in the middle and lower thirds of D1. The loss in the post-posterolateral group has in places gone on to complete devastation.

Presented in tabular form, the figures are as follows:¹

	Postero-lateral	Post-posterolateral
In upper third of C8 smaller counts on right in	.72	.60
In middle third of C8 smaller counts on right in	.50	.15
In lower third of C8 smaller counts on right in	.79	.59
In upper third of D1 smaller counts on right in	.33	1.00
In middle third of D1 smaller counts on right in	.20	.67
In lower third of D1 smaller counts on right in	—	.67

¹ Each of these percentages represents 15 to 20 counts on separate sections.

It is seen that the maximum of the lesion in the two groups falls at different levels, i. e., for the posterolateral at the lower third of C8 and for the post-posterolateral in the upper third of D1.

A thinning is apparent in a number of sections in the central group on the right, and also in the substantia gelatinosa.

The microscopic changes are of the same character as described above. The nerve cells in all groups show various degrees of degeneration and are occasionally surrounded by satellites, some of which suggest neurophages. Perivascular infiltration is of the same focal type as at higher levels, and is not more marked here than elsewhere. The neuroglia is not notably increased either in the gray or the white matter. The pia is slightly thickened in a few sections at the point of exit of the anterior roots, but as a rule appears normal except about the infiltrated vessels in the anterior fissure. The white matter appears normal. In the lower third of D1, the difference in the size of the horns becomes less marked on account of the disappearance of the posterolateral group. The difference in the post-posterolateral groups is maintained throughout the segment. Atrophic cells are found, but no perivascular infiltration. By the time the second dorsal is reached the abnormal cells have disappeared. In two sections from the lower dorsal region the nerve cells all stain well and are present in equal numbers on the two sides.

A section from the third lumbar shows conditions resembling those in the cervical enlargement, but of a mild character. There is a sprinkling of lymphocytes about the vessels in the anterior fissure, hemorrhages and hyaloid droplets in the gray matter, a few atrophic cells, a few others showing the axonal reaction, and clusters of satellites about some of the smaller nerve cells in the centers of the anterior horns. There is, however, no apparent cell loss.

Sections stained to bring out the neuroglia show at the cervical, dorsal, and lumbar levels, a moderate thickening of the peripheral glia with prolongations into the white matter, and also considerable perivascular gliosis. No gliosis in the gray matter.

The peripheral nerves were not separately examined but both anterior and posterior roots, as they are present in various sections appear normal.

Discussion.—The present case undoubtedly belongs to the group described by Pierre Marie and Foix, but represents a much less advanced stage of the lesion than is found in their histological cases. In their illustrations the nerve cells on the affected side have completely disappeared at the maximum of the lesion, and the anterior horn is reduced to a linear cicatrix. The present

case is at a stage in which it is possible to determine the cell groups chiefly affected.

Unfortunately the etiology of the atrophy in this case cannot be settled owing to the absence of a Wassermann reaction. The perivascular lymphocytosis which is present in both brain and cord, is of course suggestive, but it is very mild, and the picture is certainly not the usual one of cerebrospinal syphilis. As to the theory of a progressive ischemia of arteriosclerotic origin, it must be said that the sclerotic changes are not very advanced in the vessels either of the viscera or of the central nervous system. The history of prolonged alcoholism points to a possible toxic origin, which is at least consistent with the perivascular gliosis in brain and cord, the peripheral gliosis, satellitosis and perivascular accumulations of pigment.

A point of importance in the pathology of the cord lesion is that affection of the nerve cells and perivascular infiltration are more widespread than would appear from the perceptible muscular atrophy. As has been pointed out, affected nerve cells occur in all groups on both sides and are present at levels above and below those at which cell loss can be demonstrated, and they also occur in the lumbosacral cord. Perivascular infiltration disappears in the dorsal region to reappear in the lumbosacral. This extensive involvement of the nerve cells links the condition with chronic anterior poliomyelitis (progressive muscular atrophy). In fact, in view of the prominent and widespread cellular lesions, the perivascular exudation and the changes in the lumbosacral cord, which apparently denote beginning involvement, we fail to see fundamental differences in the pathology of the two conditions, although of course clinically they are dissimilar. Marie and Foix, however, found that such nerve cells as were present appeared normal, and consider that this fact constitutes an essential difference between anterior tephromalacia and chronic anterior poliomyelitis.

The reason for the early and advanced involvement of the posterolateral and post-posterolateral groups of the cervical cord is not clear. The theory of a vascular distribution of the lesion was suggested by Marie and Foix. Perivascular exudate in the present case is limited to the sulcal branches of the anterior spinal arteries, and their branches to the anterior horns. The posterior

spinal arteries show no trace of exudate. A branch of the sulcal artery passes obliquely across the anterior horn, separating the posterolateral and post-posterolateral groups, but this vessel often shows no infiltration and indeed is less affected than the more anterior branches. It may be that the posterior groups are less well supplied with blood than the mesial and anterolateral groups.

Other factors than the vascular may also be considered in explanation of the selectivity of the lesion. In the first place, the neurones supplying the muscles of the hands are the longest in the upper extremity and would therefore presumably be early affected in any disease process.

Secondly, both phylo- and ontogenetic considerations may be of importance. The small muscles of the hand are a late development phylogenetically, are among the last of the upper extremity to be differentiated in the embryo, and are among the last, if not the last, muscles over which voluntary control is acquired.

It may be objected that the nerve fibers to the intrinsic muscles of the feet are longer than those to the hands, and that the latter develop earlier in embryonic life than the former.¹⁰ It seems probable, however, that the great excess of exercise which the hand muscles receive, as compared with those of the feet, would outweigh these considerations and account for the earlier involvement of the hand muscles.

An additional possibility may be mentioned, which is that a primary disease of the peripheral nerve fibers might conceivably lead to entire destruction of their cells of origin. Alcohol or some other agent producing a peripheral neuritis might for unknown reasons select these particular fibers.

Finally, the localized muscular atrophy and the differential cell loss in the cord permit one to draw conclusions as to the distribution of the cell groups for the small muscles of the hands. The present case is a demonstration from human pathology that these groups are situated in the posterolateral and especially in the post-posterolateral columns, extending from the middle of the seventh cervical through the first dorsal segment. It does not seem possible on the basis of the present case to go farther and attempt to distinguish groups for the thenar, hypothenar and interossei muscles. The gaps on the left (on which there was atrophy of

the thenar and hypothenar muscles but not of the interossei) are situated as a rule at the angle between the anterior and posterior horns, while on the right (on which there was in addition atrophy of the interossei) they occur both at the angle, along the posterior edge of the horn and at the posterolateral angle. Nevertheless, the findings are not sufficiently different on the two sides to justify definite conclusions. It is probable also that the cells of origin of the median and ulnar are commingled.

Summary.—A case of isolated atrophy of the small muscles of the hands, of uncertain etiology in a 58-year-old man of alcoholic habits. The essentials of the lesion in the cord are:

1. Atrophic and degenerative changes in the nerve cells, widely distributed in the gray matter, but more prominent in the posterolateral and post-posterolateral columns.
2. Cell losses in the posterolateral and post-posterolateral columns.
3. Vascular changes, which consist in a thickening and a mild perivascular lymphocytic infiltration, the latter being confined almost entirely to the sulcal arteries and their branches to the anterior horns.
4. A characteristic distribution, the lesion commencing in the middle of the seventh cervical and extending through the first dorsal segment.

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CEREBELLAR SYNDROME*

By W. F. SCHALLER, M.D.

The subject which I have chosen, the symptomatology of cerebellar disease, is one that is alike of surpassing interest to the aurist, the oculist and the neurologist. The neurologist today must understand something of the functional ear tests as well as be able to handle an ophthalmoscope, and it is equally important for the man in special lines of work to know something of the ordinary neurological examination. As an introduction to my subject I will briefly mention some of our later notions as to the anatomy and physiology of the cerebellum. Edinger (1) who has studied this part of the brain from a phylogenetic point of view calls attention to the fact that the vermis first appears in the lower forms of animal life and that it is essentially the organ for what he calls the statotonus, that is the coördinated muscular tension stimulated by impulses originating in the labyrinth and in the muscles and joints. The cerebellar hemispheres which appear in

FIG. 1. Schematic representation of the principal cerebellar connections, modified after diagrams by Moritz and by Thomas. There are two main cerebellar systems:—

1. Cerebro-cerebellar system. This pathway of fibers, interrupted by numerous neurones begins in the posterior columns of the cord, passes by way of the fillet to the thalamus, cerebral cortex, middle peduncle of the cerebellum, cerebellar cortex, dentate nucleus and superior peduncle to the motor tegmental nuclei—red nucleus, Deiters. From this point peripheral connections are made, one of these being the rubro-spinal tract. This system has for its function regulation of muscular movements such as is represented by the finer synergic movements of the extremities. The cerebellar connections to the motor cranial nerves are indicated by the tract from Deiters' nucleus to the posterior longitudinal bundle.

2. Cerebello-bulbo-spinal system. Stimuli from the spinal cord by way of the afferent cerebellar tracts, Gowers' tract, direct cerebellar tract, and from the vestibular apparatus by way of the vestibular nerve reach the vermis and from here are carried to the tegmental nuclei and then to the motor spinal tracts. The vestibular-spinal tract from Deiters' nucleus illustrates an important centrifugal spinal connection in this system which has to do with the equilibrium of the body.

The vestibular-ocular reflex follows the course: Labyrinth, vestibular nerve, Deiters' nucleus, posterior longitudinal bundle and oculo-motor nuclei.

* From the Neurological Clinic, School of Medicine, Leland Stanford Jr. University, San Francisco, California. Read before the Pacific Coast Oto-Ophthalmological Society, at Seattle, Washington, July 2, 1914.

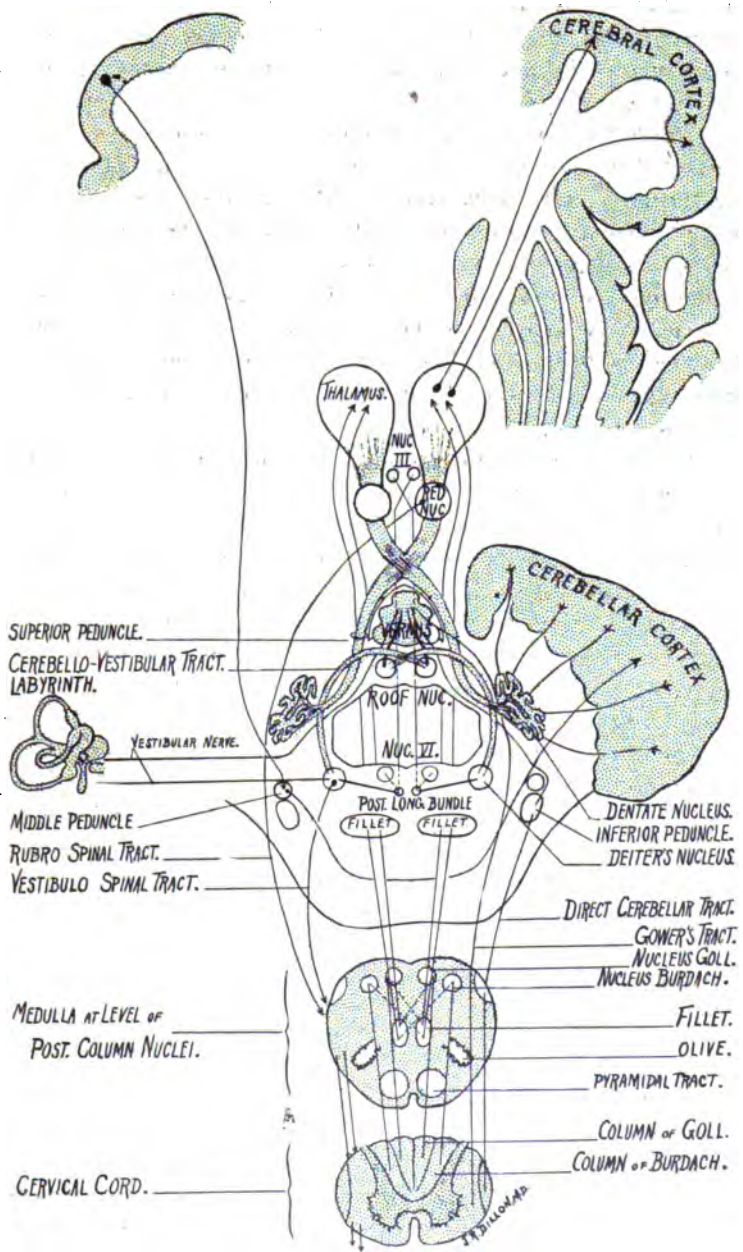


FIG. 1.

the higher forms have to do with the finer synergic movements of the extremities, the disturbance of which is shown by true ataxia. According to Edinger a peripheral stimulus originating for instance in the fibers of the posterior columns reaches the thalamus and finally the cerebral hemispheres. From here this stimulus is translated into a motor impulse by way of cortifugal fibers entering the pons and finally reaches the Purkinje cells in the cerebellar cortex. These principal pathways are illustrated in Fig. 1. Edinger therefore believes the cerebellum to be an organ regulating the muscular tonus. He quotes Sherrington in the experiment of separating the midbrain from the cerebellum by dividing the tecto-cerebellar tract and thereby producing decerebrate rigidity. This would indicate that the cerebellum constantly receives inhibition from the cerebrum.

Rothmann and Bárány, two noted authorities, have recently described centers in the cortex of the cerebellum. The latter speaks of tonus centers and would appear to incline towards Edinger's theory while Rothmann inclines to the view of definite cortical localization such as we have in the cerebral cortex, and by electrical stimulation claims to have produced muscular contractions in the corresponding members. His conclusions differ from those of Horsley, however, who has experimented previously along similar lines.

The title of this paper, "Cerebellar Syndrome," is rather comprehensive and it was chosen for the lack of a better one. I shall at times digress somewhat from my subject and speak of the pontine region and vestibular apparatus as anatomically and physiologically these structures are so closely related. The pathological processes producing cerebellar symptoms are very commonly new growths, abscesses and vascular lesions, more rarely atrophy and chronic degenerative and chronic inflammatory processes.

CEREBELLAR ATAXIA

Cerebellar ataxia differs from the ordinary ataxia as seen in tabes, and presents distinguishing features which differ from other muscular incoördinations. The gait in cerebellar disease is characteristic—a staggering gait resembling that of a drunken person. In the Romberg test closure of the eyes does not affect the swaying as in tabes. In ordinary tests for ataxia of the extremities

such as the finger to nose test, the tabetic with the eyes closed cannot touch his nose because he does not know its exact location. Under the same conditions the individual with cerebellar disease knows exactly the location of his nose but cannot accurately coordinate his different movements and thus passes the mark. Thomas has named this symptom dysmetria. We have seen that the cerebellum is the organ which regulates the synergic movements of the muscles of the extremities. A disassociation of these synergic movements results in asynergia. This symptom is shown in Fig 2, in which the trunk does not coordinate its move-



FIG. 2. C. H. Male, aged 9 years. Diagnosis: Brain tumor. Asynergia of trunk and lower extremities.

ments with the movements of the legs: whereas the legs progress in walking the body remains behind and projects backwards. Asynergia may also be brought out as follows: When the patient's foot is raised to touch the examiner's hand held at a short distance from the ground the thigh is flexed on the pelvis and afterwards the leg on the thigh causing a complete disassociation of the movement. Babinski (2) who described the above phenomenon also is the discoverer of another very closely related to it: *Adiadochokinesis* or the inability to rapidly execute certain movements which require the action of antagonistic muscles such as rapidly pronating and supinating the hands. The ataxia in cerebellar disease when unilateral is as a rule on the same side as the lesion.

FALLING SYMPTOM

Starr before the International Medical Congress in London last year called attention to sudden falling as an early sign of disease of this portion of the brain. A constant tendency to fall to one side or another has been noted in the Romberg test. Bárány (3) who assumes the presence of tonus centers in the cerebellum believes that there are two such centers in either half of the vermis. Either a destructive or an irritative lesion would then determine the direction of the falling; and as an irritative lesion, such as a tumor, is frequently in this region, falling to the side of the lesion predominates. When falling is dependent upon a vestibular process this falling is in certain definite relation to the direction of the concomitant nystagmus, being in the direction of the slow component of the nystagmus. When the position of the head is changed, the direction of the falling is also changed to correspond with the direction of the slow component of the nystagmus; for example if with a slow component to the left the head be turned at an angle of 90 degrees towards the right shoulder the patient will fall forwards, but if the head be turned on the contrary at an angle of 90 degrees towards the left shoulder the patient will fall backwards. In cerebellar disease however there is no relation between the direction of the nystagmus if present or produced and the direction of the falling, nor does change of position of the head change the direction of the falling. Such a condition would speak in favor of a lesion of the vermis.

SYMPTOMS GENERALLY ASSOCIATED WITH INCREASED
INTRACRANIAL PRESSURE

Again there are certain symptoms which are most frequently caused by an increase of intracranial pressure and are not strictly speaking brain tumor symptoms. I refer to headache, nausea, vomiting, vertigo and choked discs. It is admitted that focal lesions may cause any or all of these symptoms but the immediate relief of the tension of the cerebrospinal fluid by decompressive operations by puncture of the corpus callosum or of the lateral ventricles will cause a corresponding amelioration of the above symptoms. Headache in posterior fossa tumor is frequently in the occipital region and not seldom accompanied by rigidity of the neck muscles. In the cases of posterior fossa tumor which we have observed and studied the above symptoms were all present. Bárány (4) states that nausea and vomiting are rare symptoms in this region. Oppenheim (5) states that here choked disc is not an early symptom.

In tumors of the fourth ventricle, particularly cysticercus of this cavity, there occur attacks of vertigo on abrupt movements of the head. This symptom brought out by Bruns is explained by the fact that the detached daughter cysts impinging on the walls of the mother cyst cause a stimulation of the vestibular



FIG. 3. A. D. B. Male, aged 20 years. Diagnosis: Angiosarcoma in the 4th ventricle. Probable origin tela choroidea inferior.

nucleus in the floor of the 4th ventricle. In the case here reported (Fig. 3) the tumor was an angiosarcoma probably originating from the inferior tela choroidea. The severe attacks of vertigo paroxysmal in nature were without doubt due to the direct pressure of this growth on the vestibular nucleus. Of the less usual symptoms of increased intracranial pressure we may have either unilateral or bilateral anosmia due to flattening out of the olfactory nerves at the base of the brain, or optic atrophy and changes in the pattern of the visual fields from pressure of the distended third ventricle on the optic chiasm. Here we may diverge a trifle to emphasize the importance of removing a condition. When increased pressure of the cerebrospinal fluid is present which may cause such a varied and confusing picture, callosal puncture as advised by Anton of Halle may be followed by gratifying results. This we have advised in several patients. In one patient lumbar puncture, which is attended with some risk in brain tumor, brought about improvement in a greatly impaired mentality, but was not repeated on account of alarming symptoms from the heart and respiratory systems.

NYSTAGMUS

Nystagmus properly speaking is not a symptom of disturbed cerebellar function. The cerebellar apparatus is however in such close anatomical and physiological relationship with the vestibular apparatus and the posterior longitudinal bundle and Deiters' nucleus that nystagmus is often spoken of as being a cerebellar symptom. We have observed a case (Fig. 4) of cerebellar softening involving the white matter of the hemispheres and the corpus dentatum in which there had been no evidence of nystagmus in the symptomatology of the case. The studies of Neumann and Bárány however have rendered the determination of nystagmus so important an aid to diagnosis of posterior fossa conditions that a brief discussion cannot be dispensed with. The pathway of the vestibulo-ocular reflex is diagrammatically outlined in Fig. 1, assuming that we are dealing with an intracranial nystagmus such as is distinguished by a quick and slow component, by persistence under closed lids and long duration and the absence of eye conditions which may be a cause. Firstly spontaneous nystagmus: When a spontaneous nystagmus exists the direction

of this nystagmus points towards the lesion when the latter is central, *i. e.*, retrolabyrinthine not peripheral. Neumann states that this is true in approximately 80 per cent. of all lesions. The

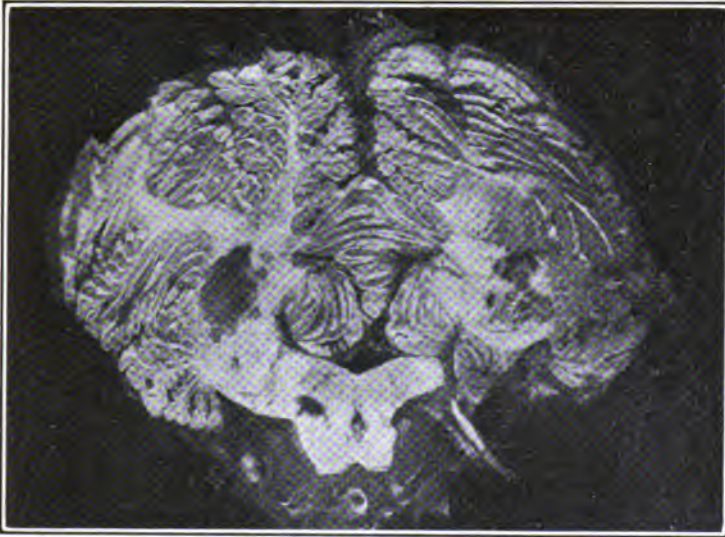


FIG. 4. J. E. S. Male, aged 57 years. Double-sided softening in white matter of cerebellar hemispheres.

vestibular nystagmus as has been mentioned consists of a slow and a quick component, the slow movement being the result of the labyrinthine stimulus and the quick component the compensatory action of the supranuclear centers. These latter were until lately supposed to be in the central cortex, but Bauer and Leidler, quoted by Marburg (6), in a series of experiments showed that removal of the cortex did not remove the quick component. The analysis of intracranial nystagmus now permits us to give a satisfactory explanation of the phenomenon of conjugate deviation of the eyes in cases of capsular hemorrhage. The inhibitions of the supranuclear centers being removed on one side by a lesion, the vestibulo-ocular reflex is given full play, resulting in the fixed lateral position of the eyes (Fig. 5). Neumann has described a case where there was inability to move the eyes laterally voluntarily but when the eyes were fixed by the finger this was rendered possible. If in this same case a nystagmus was produced by turning, a deviation conjuguée was produced.

In pontine lesions Marburg (7) gives two causes for deviation conjuguée, a cerebral and labyrinthine form: "Wir hätten

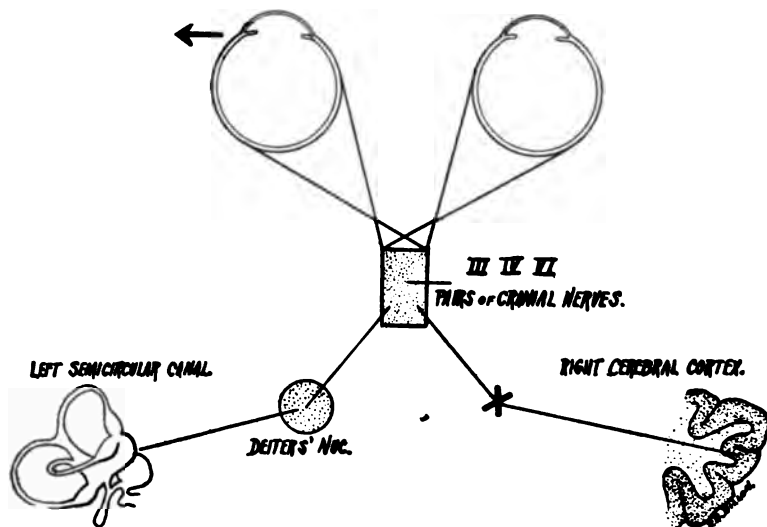


FIG. 5. Schematic Representation of Conjugate Deviation of Eyes. (X) Lesion in supranuclear center. Arrow indicates side towards which eyes deviate.

demnach bei pontinen Herden eine doppelte Ursache für die Deviation conjuguée: Die erste besteht in einer Läsion der Willkürbahnen, zerebrale, hemiplegische Form. Sie ist zumeist herdkontralateral mit gleichseitiger Hemiplegie verknüpft und dürfte ein Reizungssymptom darstellen; sie ist an Herde im Brückenfuss nahe seiner oralen Grenze gebunden, wenn diese ein wenig haubenwärts vordringen. Die zweite, die labyrinthäre oder vestibuläre Form, findet sich bei Affektionen der Brückenhaube, wenn diese den Deitersschen Kern resp. dessen System lädieren. Sie scheinen herdgleichseitig und herdkontralateral, ganz im Sinne von Bruce als Reizungs- und Lähmungssymptom auftreten zu können."

This brings us to the consideration of induced nystagmus such as may be produced by various means, by rotation, by the caloric test, by aspiration in fistula and by galvanism. A description of the physiology of these functional tests may be dispensed with and a brief mention made of a few aids to diagnosis whose worth appears to be established. When there is a question of whether

a spontaneous nystagmus is due to a lesion in the cerebellum or to a lesion in the peripheral vestibular apparatus the caloric test gives certain information (Neumann). From other symptoms the probable location of the lesion is determined and the ear on the opposite side to the lesion is syringed with cold water. If the vestibular apparatus is affected the nystagmus produced will last from 2 to 3 minutes, but if the lesion is central there will be an enduring nystagmus lasting from 8 to 9 minutes. As is well understood peripheral destruction of the vestibular apparatus or nerve will show an absence of reaction to the caloric test. In cases of cerebellar pontine angle tumor the caloric test is also absent; Bárány (8) states that in 30 cases observed by him this phenomenon has not varied.

After turning we sometimes get a certain violent reaction consisting of a prompt and marked nystagmus together with great dizziness, and falling. This has been found in tumors of the brain not involving the cerebellum, but is present on account of the probable removal of the inhibitory influence of the higher centers on the peripheral and central vestibular apparatus. This reaction is called the tumor reaction. In a recent case of a tumor involving the corpus callosum this reaction was marked (Fig. 6).

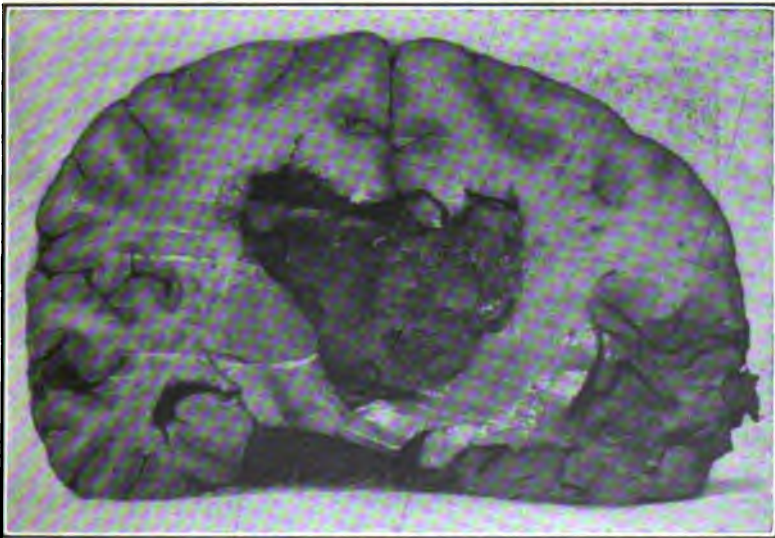


FIG. 6. K. A. Male, aged 47 years. Diagnosis: Sarcoma of tela choroidea superior involving corpus callosum.

Galvanic Tests.—Babinski has described a rotation of the head normally to the side of the anode when both positive and negative poles are placed a little above the tragus on either side and a varying current of from 1 to 10 ma. is allowed to pass through the head. This is said to be due to the electrical stimulation of the central vestibular apparatus. In case of a unilateral lesion Babinski holds that the head rotates or inclines toward the side of the lesion in man. This however has not been fully confirmed by animal experiments.

Ruttin has also employed galvanism in functional vestibular tests. To quote from an article by Dr. Harrington Graham (9): "If cold water is simultaneously run into both ears under similar conditions there should be no nystagmus. If one vestibular apparatus is less sensitive than the other there should be a nystagmus to that side. The same is true of the nerve in relation to the galvanic test. The kathode applied to both ears under like conditions should produce a nystagmus toward that side containing the more reactive nerve. Therefore, if we have a patient with vestibular symptoms, as spontaneous nystagmus or dizziness, or dizziness with forced head movements, and there is no nystagmus on applying the caloric test to both ears under like conditions, and there is a nystagmus on application of the galvanic test simultaneously to both ears, we can readily make a diagnosis of retrolabyrinth affection and determine the side that is over- or under-active. In the application of these tests one has to keep ever in mind the physiology of the vestibular tracts as in cases where the cerebellum is affected we may produce by the double caloric a nystagmus to the sound side instead of to the diseased, thus confusing our diagnosis; we must assume the presence of a tract (cerebellar) that acts as a brake on the normal impulses and when the brake is off that side will overact, producing a caloric nystagmus to the opposite side. This we see in cerebellar tumor, abscess, etc. It will be readily seen that we have in these cases of Ruttin's the means for quickly ascertaining whether either the vestibule and semicircular canals or the nerve on one side or the other is over or under sensitive or normal, thus winning one more point in our differential diagnosis."

POINTING TESTS OF BÁRÁNY

Graefe first found that in ocular paresis when a patient attempted to point to a cross on a black board with his eyes closed, he would point to one side. Neumann and Bondy discovered that when a patient was turned in a rotary chair that the objects of the external world appeared to him to be misplaced. Bárány deserves the credit of describing an objective test for the notion of position of external objects: After the vestibular apparatus has been stimulated a normal individual will point to one side of an object in space after such stimulation by various methods of turning, syringing, etc., and the direction of this error will be in the direction of the slow component of the nystagmus produced. For instance if cold water be injected into the right ear a nystagmus will be induced towards the left and pointing errors to the right. As the nystagmus varies so also will the pointing vary, and positions of the head will therefore determine the different directions of the pointing, in either the horizontal or vertical plane. These errors of pointing may be demonstrated by the extremities and also by the head; the errors are constant, irrespective of the position of the extremity, as for example there is no change if the arm be in pronation or supination. Adhering to the notion of tonus centers in the cerebellar cortex Bárány believes that there is a cerebellar localization of such centers for pointing. A lesion of the cerebellar cortex will produce anomalies of pointing which have a diagnostic significance. For instance if there be a destructive lesion in the arm center of the right cerebellar cortex the patient will no longer point to one side after turning but will point correctly. Spontaneous errors of pointing may also be present, but for Bárány the characteristic symptom of cerebellar cortical disease is the definite abnormal reaction of pointing after turning, or after the caloric tests.

Lesions of other portions of the brain may cause spontaneous errors of pointing by reaction at a distance or "*Fernwirkung*" (Bárány) (10), but in these cases the pointing after the above procedures is normal. Recently Rothmann (11) has published an interesting history of a man who, after a traumatism of the head followed by vertigo, nausea and papillary edema, not alone showed spontaneous but also induced errors in pointing. At the operation a subpial sero-sanguineous effusion was found over the

supramarginal gyrus. Following the operation this patient pointed normally. In this patient there was no sensory or motor involvement in the arms. Rothmann believes that this case speaks strongly for a cortical localization to explain the abnormal pointing reactions. Recently we have observed a patient in the Stanford Clinic who complained of nervous symptoms and who showed a well-marked astereognosis of one hand with profound involvement of the deep sensibility and very little of the superficial sensibility in this extremity. This patient was tested for pointing

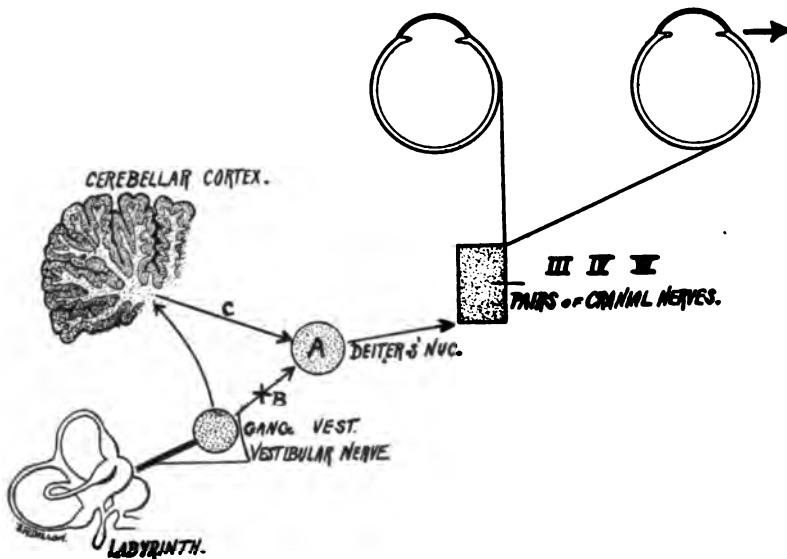


FIG. 7. Illustrating Labyrinthine Reactions after Syringing Left Ear with Cold Water. Arrow indicates direction of resulting nystagmus. A lesion in Deiters' nucleus (A) will block nystagmus and influence normal pointing tests, i. e., patient will point normally when he should deviate. A lesion in the vestibular nerve (B) between the vestibular ganglion and Deiters' nucleus will block nystagmus but will not influence normal pointing tests, i. e., patient will deviate on pointing.

and on the affected side spontaneous as well as produced errors were found on repeated tests in the neurological clinic and the ear clinic. This patient presented no signs of cerebellar disease. The inference is that in this case the pointing errors were due to a cortical lesion. In the caloric test a comparison of the behavior of the pointing and the nystagmus produced may give information as to the localization of a lesion. For instance a lesion in Deiters'

nucleus will block nystagmus but will also influence pointing tests through the cerebello-vestibular tract. A lesion of the vestibular nerve between the vestibular ganglion and Deiters' nucleus will block nystagmus but will not influence the normal pointing tests on account of undisturbed cerebellar function. Diagram 7 illustrates these points.

OTHER SYMPTOMS OF CEREBELLAR DISEASE

Scanning speech, slowness of speech, fixed attitude of the head and trembling have been described as symptoms of cerebellar disease. As in tumors elsewhere there is sometimes localized sensitiveness to percussion over the area of growth. A very interesting and curious symptom of cerebellar disease has been described by Babinski (2) and called by him cerebellar catalepsy. This was shown to perfection in our case of cerebellar softening previously mentioned (Fig. 4). This man who could not stand without support, could while reclining on his back hold both legs practically immovable above the trunk and free from the bed; indeed he could perform this maneuver as well if not better than the normal individual and without as much fatigue. Other symptoms referable to the lower extremities are not infrequently found in tumors of the posterior fossæ due perhaps again to increased intraspinal pressure and these are: Tetany of the muscles of the legs and radicular pain, and absence of the knee jerks. Contralateral pressure of a tumor on the pyramidal tract may, according to Oppenheim, give the Babinski sign.

SYMPTOMATOLOGY OF CEREBELLO-PONTINE ANGLE TUMOR

In tumor of the posterior fossa situated at the cerebello-pontine angle we often have a combination of symptoms readily explained on anatomical and physiological grounds. These symptoms are unilateral facial neuralgia, facial nerve paresis, corneal anesthesia, nerve deafness, absence of caloric reaction on the side of the lesion, together with some or all of the signs of intracranial pressure mentioned above. Abnormal pointing reactions have been noted in the upper extremity on the side of the tumor and are explained by pressure of the tumor on the arm center in the cerebellar hemisphere. In one case of cerebello-pontine angle tumor which was successfully removed by Dr. Emmet Rixford,

the first symptom was difficulty of hearing over the telephone in the left ear. This patient would often suffer from dizziness on getting out of bed. Failing eyesight finally brought the patient to the clinic and a marked papillary edema was found. The ear symptoms, corneal anesthesia and slight facial paresis made the diagnosis most probable, but in this case the labyrinth acted normally to the caloric test on repeated examination. This experience was in striking contrast to the absence of excitability of the labyrinth in Bárány's (8) cases.

BÁRÁNY'S SYMPTOM COMPLEX

Bárány (12) has described a group of symptoms brought about by a circumscribed increase of fluid pressure in the cisterna pontis lateralis, at the cerebello-pontine angle, due to adhesions and consequent interference with the cerebrospinal fluid circulation. Otitis media, syphilis, rheumatism and influenza may cause this condition. The symptoms are difficulty in hearing, vertigo, ringing in the ears, occipital headache and pointing errors outward on the affected side. Variations in acuity of hearing are characteristic. Babinski (13) has previously recommended lumbar puncture and the withdrawal of a considerable amount of fluid for the relief of these symptoms. This procedure evidently acts by breaking up adhesions of the meninges. Bárány has practiced trephining over the mastoid process in several cases.

SUMMARY

1. It is impossible to state that any one of the above described symptoms is absolutely dependable in cerebellar disease, therefore the classification cerebellar syndrome.
2. Of the more dependable symptoms we would class ataxia of cerebellar type, asynergia, adiadochokinesis and cerebellar cataplexy; falling symptom, and variations from the normal in the functional labyrinth tests and the pointing reactions of Bárány.
3. Less dependable symptoms are those produced by intracranial pressure: Nausea, vomiting, vertigo, choked discs and nystagmus.*

* I wish to here make acknowledgment to Dr. James R. Dillon, assistant in the neurological clinic, for drawing the diagrams illustrating this article and to Dr. Harrington B. Graham, of the ear, nose and throat clinic for the special ear examinations in certain of the cases reported, also for his kind advice and criticism in the preparation of that part of this paper which deals more particularly with the labyrinthine reactions.

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AN ANGIOMA OF THE CEREBELLUM

BY L. NEWMARK, M.D.

SAN FRANCISCO

The tumor which is illustrated here by the photograph of a section stained according to the method of Van Gieson is an object of some interest because its effect, which was fatal, seemed out of proportion to its size, which was very small, and still more because its histological examination showed it to be of a kind very rarely met with in the cerebellum, where it was found. An inquiry into the literature of the subject furnished a moderate number of reports of cases of circumscribed angioma in the cerebral hemispheres, several of the writers remarking upon the infrequency of such an occurrence. But of a circumscribed angioma of the cerebellum only what looks like a solitary instance was found recorded in a brief abstract of a Russian article, where it is stated that a "hemangioma in the cerebellum" was successfully removed from a girl nine years old.¹

The patient from whom my specimen was obtained was a woman, aged 32 years. About a year and a half before coming under my observation she had had a first attack of pain in the back of the head, which lasted for a short time only. A month later she suffered again in the same way for a few days; and two months after the second attack a third, severe, one occurred. Vomiting did not accompany these headaches. There followed a period of ease which continued until six weeks before I first saw her. Then the occipital headaches recurred: intermittent at first, but persistent for the past month, and, when severe, attended with vomiting and with diplopia. She had taken to her bed three weeks before I became acquainted with her. She is said to have complained of dizziness, but no statement was obtained indicating a disturbance like cerebellar staggering. Buzzing in the left ear was mentioned as having occurred at one time. Of late she would have attacks in which consciousness seemed to be obscured, she could not speak, would perspire freely and turn yellowish. Such attacks would last a few minutes and would be followed by vomiting. In the course of her six weeks' sickness she had been sub-

¹ Journal of the American Medical Association, May 9, 1914.

jected to treatment with iodide of potash, mercury and finally salvarsan.

The patient suffered in the manner described during the week she was under our observation: there were occipital headaches, attacks of altered consciousness and vomiting. She was very emaciated. The optic discs were only very slightly blurred; in fact I was not sure of this and had an oculist examine her repeatedly; he never could convince himself that there was any choking of the disc and considered what he did see as rather indicating a toxic neuroretinitis. There was nystagmus on looking to either side, rather more marked on looking to the right, but none on looking upward or downward. The pupils reacted well to light. The hearing was unaffected; but on account of her frequent vomiting and her apparent prostration Bárány's caloric tests were not made. There were no disturbances of sensibility anywhere until the last day of her life, when it was noted that the sensibility of the right cornea was dulled and from the left no reflex at all was obtained. The cranial nerves were not otherwise affected, nor was the deglutition impaired. The headaches were not felt more in one half of the occiput than in the other, radiographs disclosed nothing abnormal, and careful and repeated testing of the skull elicited no tenderness to percussion or pressure anywhere, the back of the head being most diligently examined in this respect. There was no ataxia of the extremities, no adiadochokinesis, no astereognosis. She seemed too feeble to be taken from her bed and tested as to her gait, but on the morning of the day on which she died she surprised us by the ease and vigor with which she rose from the recumbent into the sitting posture.

The knee-jerks as well as the reflexes of the upper extremities were constantly absent, while the Achilles reflexes could at all times be easily elicited.

On the afternoon of December 10, 1913, the husband had just assented to the proposal to explore the cerebellum when word came from the hospital that the patient was worse, and she soon passed away.

The contents of the cranial cavity having been removed a limited, cyst-like, collection of fluid in the pia-arachnoid drew attention to its site at about the middle of the posterior margin of the left cerebellar hemisphere. A horizontal incision into the cerebellum at this place revealed a dark-red spherical tumor, about the size of a pea. The incision had left the globule intact; part of the little sphere was free, and part seemed embedded in the surrounding tissue. The tumor did not quite reach the surface of the cerebellum, but was a few millimeters beneath it.

The whole tumor with adjacent tissue was excised in one small block. Sections were submitted to Professor Ophüls, the pathologist of Stanford University, who, with accustomed generosity, examined them and sent the following report: "Small tumor of

the cerebellum, about five mm. in largest diameter. The tumor is made up almost altogether of capillary bloodvessels of various sizes. Some of them are narrow and cylindrical, others very large and sinuous. Many of the cylindrical capillaries are in a state of collapse. Little, fairly cellular, loosely constructed fibrous tissue between the bloodvessels. The tumor is intimately connected with the pia mater, well circumscribed. It has caused some destruction in adjoining cerebellar tissue by pressure, but does not extend into brain tissue proper. Angioma of the cerebellum."

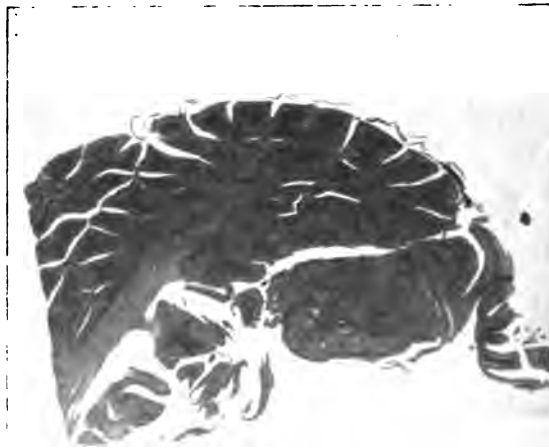


FIG. 1

To this may be added that no hemorrhages had occurred in or around the tumor; nor were thromboses or calcified vessels seen. In the center of the sections the vessels are seen to occupy the field more exclusively than at the periphery, where there is more tissue between them, so that one gets the impression of a cavernous structure. But there are not the large spaces filled with blood and separated by walls of connective tissue and lined with endothelium, which constitute the cavernous angioma.

There was no considerable degree of hydrocephalus. Macroscopic and microscopic sections from other parts of great and small brain showed no further lesion. In sections of the meninges, where the fluid had collected, nothing more abnormal was noticed than a slight widening of the space between their layers.

The autopsy could not be extended beyond the skull.

The antecedent occipital headaches of more than a year before may be regarded as premonitory. In the final phase of seven weeks' duration the course did not seem to differ much from what is observed in cases of less uncommon tumor. However, considering the severity and frequency of the headaches and the vomiting and the attacks of altered consciousness together with the loss of knee-jerks which seemed attributable to increased pressure in the cerebro-spinal canal one would have expected more swelling of the optic discs. It was on account of the assumption of a tumor in the posterior fossa with much pressure that we forbore to practise the lumbar puncture, fearing the consequences under such conditions.

Had the operation been performed it is not improbable that the collection of fluid at the site of the angioma would have made the impression of a circumscribed serous meningitis and that the operation would have concluded with the evacuation of the fluid, and the little tumor have been overlooked and allowed to remain. But even from so limited an intervention benefit might have ensued, for the local accumulation of fluid may have been the immediate cause of the suffering and death rather than the tumor itself.

Lacking reports of other cases of tumor of like structure in the cerebellum I can only cite for comparison accounts of circumscribed angiomas which have been observed in the cerebral hemispheres, disregarding the diffuse teleangiectatic tumors of the pia mater. Oliver and Williams² reported the removal of a "simple angioma, rather a rare form of growth apart from sarcoma." There was optic neuritis together with the other common signs of a cerebral tumor. In four other cases, of which two have been described by Astwazaturoff,³ one by Struppler⁴ and one by W. J. Sweasy Powers,⁵ the tumors were cavernous angiomas, three of them situated superficially and connected apparently with the pia, while the fourth, that described by Powers, was well down in the centrum ovale of the frontal lobe and not connected with the pia mater. In none of these is it clear that choked disc was present; in none was a tumor diagnosed. Epileptiform convulsions were

² British Medical Journal, 1898, Vol. 26, p. 1267.

³ Frankfurter Zeitschrift für Pathologie, Bd. 4, and Monatsschrift für Psychiatrie und Neurologie, Vol. 29.

⁴ Münchener. med. Wochenschrift, 1900, p. 1267.

⁵ Zeitschrift für die gesamte Neurologie, 1913, Vol. 16, p. 487.

common to all of them. All the patients were women. Of these Struppler's patient interests us particularly on account of the diminutiveness of the fatal angioma. It is described as being "somewhat larger than a pea." and "hardly the size of a small hazelnut." It was situated at the upper end of the Rolandic region, accordingly remote from respiratory and cardiac centers, differing in this respect from a cerebellar growth. Yet the patient, who had been considered well only a few days before, died after a numerous series of convulsions which had begun locally and later became general. The author was struck, like myself, by the apparent disparity between cause and effect.

From the history of the occurrence of headaches in my patient about a year and a half before the final stage of the disease it may be inferred that the tumor was at least of that long standing; and from its smallness after the lapse of such a period it may be inferred that it had very little, if any, tendency to grow, and that it was capable of producing its evil effects only by congestion, edema or hemorrhage.

Perhaps the angioma was congenital. In connection with this suggestion reference may be made to an interesting article by Oppenheim,⁶ who has seen a number of cases following a peculiar course which he is inclined to ascribe to a congenital "Kavernom" in the brain. A "cavernoma" in the sense in which the term is used by Oppenheim would seem to include all varieties of hemangioma. The coexistence of an angioma of the skin in the patient, or even in a parent of the patient, is held to afford support to the diagnosis. Oppenheim quotes von Bergmann as stating that the "Kavernom" is rare, but he himself thinks that the angioma cerebri cannot be such a rare tumor if the conception of it be broad enough, and that it has surely generally eluded clinical, and perhaps often escaped anatomical, diagnosis. I had not Oppenheim's article in mind while observing the patient, and some little angioma of the skin may not have been observed. I can only say that none was conspicuous. Oppenheim considers it remarkable that in nearly all his cases the patients were males. It appears equally remarkable that in mine and in all the others I have adduced the patients were females.

⁶ Ueber klinische Eigentümlichkeiten kongenitaler Hirngeschwülste, *Neurologisches Centralblatt*, 1913, No. 1

Society Proceedings

NEW YORK NEUROLOGICAL SOCIETY

JANUARY 5, 1915

The President, DR. SMITH ELY JELLIFFE, in the Chair

A CASE OF EPILEPSY, COMPLICATED BY A TRAUMATIC NEUROSIS

By William Steinach, M.D.

The patient had presented himself at the neurological clinic of the University, with a letter from his family physician, asking that a skiagram be taken and suggesting the possibility of operative intervention. He was an assistant shipping clerk, twenty-two years of age, single; born in the United States of Irish parents, the eldest of seven children, one of whom died in infancy. All the living children, except the patient, were healthy.

Family History.—Practically negative. A maternal grandmother died of apoplexy; a sister, of spinal meningitis at the age of six months. The father died of pulmonary tuberculosis; a paternal aunt, of dropsy.

Previous History.—The patient was born after a difficult labor of fifteen hours' duration, with instrumental intervention. He was brought up on prepared foods. A slight backwardness in learning to talk and walk, and a tendency to stammer while learning to speak, were the only unusual features of his infancy. A severe attack of cholera infantum, which lasted two months, during his first year, and mild attacks of the usual diseases of childhood were his only illnesses before the beginning of the present trouble.

History of Present Trouble.—When ten years old the patient was hit on the head with a baseball bat over the left temple. The wound bled profusely, but the patient did not lose consciousness.

Two years after the injury the patient had the first convulsion. He was found in bed by his mother, jerking violently. During the convulsion, which was general in character, he passed his water and lost consciousness completely. Fifteen minutes later he had regained consciousness and was able to go to school. For four months he received treatment at the Presbyterian Hospital, and in that time had no attacks. When the treatment was discontinued, however, the same trouble returned.

The attacks, of grand mal type, were described as occurring chiefly at night. In the course of them the patient would bite his tongue and void urine. At times he would vomit immediately afterward. The attacks recurred at intervals of from three to six months. There had been, however, as many as three in one day. There were no attacks of petit mal.

Two years ago the patient was struck on the top of the head while coming out of an elevator in a bank. The accident was rather trivial.

and did not affect the patient until there was some talk of a suit against the bank, which was subsequently settled. Since that time, the patient began to have peculiar attacks, in addition to the previously described type, in which his left leg was drawn up and adducted, and his left hand flexed. These attacks were not accompanied by loss of consciousness. They became very frequent,—sometimes as many as five or six in one day, each lasting about two minutes. Dr. Steinach stated that he witnessed one of them, and that the convulsion was clonic in character, involving the left leg and hand, with the back arched in a typically opisthotonos fashion.

Physical examination showed the left pupil a trifle larger than the right; both responded to light and accommodation. There was no abnormality of the motility or fields of the eyes. The left knee jerk was a trifle more active than the right. No clonus; no Romberg; no ataxia. No sensory disturbance; no astereognosis; no asynergism.

The question in connection with this case, Dr. Steinach stated, was whether this was a case of Jacksonian epilepsy in which the convulsions were attributable to a *right sided lesion from contre-coup*, or a case of epilepsy upon which were engrafted hysteroid attacks as an expression of a traumatic neurosis.

In a recent number of the Journal of the American Medical Association Dr. Burr reported a case of multiple neuritis complicated with hysteria, and Dr. Steinach had thought the case presented of sufficient interest to be reported, in that it might be considered a case of epilepsy, complicated by a traumatic neurosis.

Furthermore, the psychogenesis of the traumatic neurosis superadded to the epilepsy was very *distinctly* illustrated. The underlying unstable neurotic disposition accompanying the epilepsy was a ready soil for the neurosis. During the ten years his major attacks had lasted, he was repeatedly questioned by the various physicians whom he consulted, about blows to the head, as being a possible cause for his epilepsy. When he did receive the blow on the head, the repeated questioning having suggested the causal relationship to convulsive attacks, they promptly appeared in the left arm and leg, showing their purely hysterical character.

A CASE OF PERIODIC PARALYSIS

By C. E. Atwood, M.D.

The case, Dr. Atwood explained, was presented on account of its extreme rarity. Fewer than a hundred cases had thus far been recorded. A careful study of the disease were first made by Westphal in 1885, and later Goldflam gave a full description of it. The text-books scarcely mention the disease.

The patient was the tenth case in a family whose history was studied for four generations. The cases were all on the maternal side, and all were born in Russian Poland.

The patient was twenty-one years of age, and the second of three children, all males. One brother and the mother were affected by the same disease, and had also been examined by Dr. Atwood. The next nearest relative was a maternal uncle. Most of the other cases were cousins, once or twice removed. One second cousin choked to death dur-

ing an attack. He had been left alone and was unable to turn his head in order to throw off some food which had regurgitated. Another second cousin, who was subject to the disease, while serving in the Russian army had a severe attack, and died from burns inflicted on him during it because he was thought to be malingering.

The medical history of the case presented, aside from the attacks of periodic paralysis, was negative. The patient was well-built, muscular, with massive quadriceps. There was no dystrophy although an associated dystrophy had been found to occur in a few of the cases on record. There were no evidences of local wasting, or local paralysis. Examination between attacks showed that sensations to touch, temperature, pain and pressure were present. The reflexes were present and normal; the cranial nerves were normal; the fundus oculi was normal; the heart and lungs, normal. There was no spasticity or flaccidity of muscle. The electric reactions were present. There were no hysterical stigmata. In all respects, physically and mentally, the patient seemed normal. His history, however, showed that he was subject to attacks of motor paralysis at intervals of a few days to a few weeks, lasting for a few hours to two or three days.

Dr. Atwood stated that he had seen the patient and examined him during three of these attacks. The first one which the patient ever had occurred when he was four years of age. It lasted twenty-four hours and was general. The second attack was at the age of twelve, and lasted twenty-four hours; also general. The third attack occurred at fourteen and was typical and severe, and was followed by milder attacks as often as once a week, or even daily. Since the age of fourteen his severe attacks have occurred, at first, one in two or three weeks; later on, once in two months; and, under treatment since 1911, have recurred at longer intervals, until lately they have increased in frequency. They are more frequent in cold weather. They are sometimes preceded by a heavy feeling in the legs, which then enables the patient to foretell an attack. Attacks usually came on at night.

The first one witnessed by Dr. Atwood was on November 14, 1911, twelve hours after its inception. It was not a very severe one, and may have been lessened in severity by magnesia citrate and an enema. The patient was entirely conscious, and no abnormal mental symptoms were noticed. The left side was more affected than the right. There was great flaccidity. The patient felt weak in the ankles, knees and wrists. He was able to get out of bed with assistance at the time of the visit, but felt weak, dizzy and faint while in the erect posture, and could not stand unsupported. Sensation to touch, temperature, pain and pressure was everywhere preserved. Muscle irritability was diminished on the less affected and absent on the more affected side. The cranial nerves were free. The left knee jerks could not be elicited; the right was barely present. The left plantar reflex was absent; the right normal. A bruit could be distinctly heard over the apex of the heart. This bruit was not present when the patient was examined between attacks. The apex beat was at the sixth intercostal space. The attack lasted twenty hours. The next day on examination both knee jerks were present and there was complete restoration in muscle function and reactions. The mitral bruit had disappeared, but the apex beat remained in the sixth intercostal space.

On December 7, 1911, an examination of the patient was made during an attack, fourteen and one half hours after it began. The patient could talk, but was otherwise almost completely paralyzed. He had eaten heart-

ily just before retiring, and had smoked more than usual. Also, the evening before, his legs had seemed weak in walking, and he could not extend the fingers of the left hand. It is possible that these symptoms may have indicated a slight attack at that time. He went to sleep as usual, however, but awoke at two a. m., unable to move the arms and hands and right leg, but could move the left leg and foot slightly. He was able to sleep again, but awoke at three a. m. and found that he could move only his head. On examination at 4:30 p. m. recovery had begun. He could move the great toes, and flex the fingers; could slightly roll the head from side to side, and slightly move the left leg and the right forearm. His paralysis was flaccid. The knee jerks were absent; ankle jerks barely present; abdominal and cremasteric reflexes present, slight. The heart showed a mitral bruit. The breathing was shallower than normal and carried on mostly by the diaphragm. The voice was fairly strong; the cranial nerves were free; there was an absence of muscle tone; no responses to direct tapping. All sensibility was unimpaired; the mind was unaffected; the mucous membranes appeared normal; the hands and feet were warm; the patient was able to swallow a glass of milk and retain it. Improvement continued from the periphery upward,—the wrists, elbows, shoulders and ankles improved during a period of about two hours. The condition returned, however, at midnight after the examination, and the patient became again paralyzed, except in the fingers and neck. He vomited at eleven p. m. the milk taken, and voided urine. He was unable to expel two enemata of soapsuds administered during the attack. The following morning he awoke at six a. m., entirely free from symptoms.

Urinalysis during attack showed increased acidity; increased indican; little albumin; increased sulphate partition. Bacteriological study of the feces showed a marked intestinal infection and a state of fermento-putrefaction, due to a clean-cut infection of the *Bacillus aerogenes capsulatus*. The same condition was found in the mother and brother in a lesser degree, and was present very slightly in the father, who was not affected.

Dr. L. Pierce Clark said that he would like to ask Dr. Atwood if he had found out whether there were times when after indulging in these various irregularities of diet the patients did not have attacks in consequence, and also if experiments had been made at different times with a view to suggesting a suitable diet and finding out just how susceptible the patients were to these changes.

Dr. Atwood, in reply to Dr. Clark's question, stated that he had not been able to handle the case closely enough to do work along such lines; the conditions surrounding the patient could not be controlled to this extent. John Mitchell, Edsall and Flexner carried out a series of such experiments some years ago, and found diminished kreatinin excretion.

There have been but few autopsies and no histological studies after death. Findings based on the urine, feces and blood are conflicting. There has been no examination of the spinal fluid, as yet, during an attack. Determining causes of individual attacks have been ascribed to muscular exertion, indiscretions in diet, exposure to cold, constipation, nervous and mental fatigue, worry and emotional excitement, etc. There is a general consensus of opinion, however, that as regards the pathogenesis of attacks a condition of auto-intoxication is present. Nearly all the various other theories are found untenable. There seems to be a

deficiency of elimination of nitrogenous metabolic products, and the absorption of some toxic substance, perhaps from the gastro-intestinal tract, which is capable of acting as a paralyzant to both the endings of the peripheral motor nerves and to the muscle-fibers themselves.

A MULTIPLE SCLEROSIS IN MOTHER AND SON

By I. Abrahamson, M.D.

Before presenting the patients, Dr. Abrahamson read a short account which the son had written of his own case, and which gave evidence of the unimpaired intelligence of the patient.

The son, a student, twenty years of age, was born in England. His earliest recollection of the trouble, he stated, dated back to four years ago, when there began to be a numbness in the limbs; weakness in the legs; tremor of the hands, together with an inability to hold objects. There were no urinary symptoms with this first attack. The trouble continued for two weeks before the patient recovered.

The recent exacerbation came on three and a half months ago, when the patient began to have difficulty in starting the stream of urine, though no urgency. Also, he was troubled with obstinate constipation. About six weeks before, on his return from the country, where he had been able to do hard work, and had had a record of walking ten miles daily, he began to notice a weakness in the right leg, which increased until he was compelled to drag it after him as he walked, and for a week's time he was unable to bend it at all. Shortly afterward his right hand became so weak that he was unable to write. There was no tremor no paresthesia, nor headaches.

It was after the second attack that the patient was examined at Mt. Sinai Hospital. The following are the findings:

Pupils normal, nystagmus to the left; bitemporal pallor of the discs; jaw jerk lively; lower abdominal and right cremasteric reflexes absent; upper abdominal reflexes diminished; all tendon reflexes lively; ankle clonus present; bilateral Babinski; fine tremor of the hands; head tremor; Romberg; spastic ataxic gait, with a tendency to go to the right. Wassermann negative; cerebro-spinal fluid negative. Increased blood pressure.

The patient left Mt. Sinai Hospital improved, but recently had another slight attack. The viscera were normal, there were no abnormalities of emotional control, no real intention tremor, no pigmentation.

The findings, together with the history of remissions and exacerbations, fully justified the diagnosis of multiple sclerosis.

The mother was Russian, and had lived for a time in England. She was 48; had eight living children, and had had no miscarriages. With the exception of the one son, all the children were healthy.

Up to her twenty-second year the patient had been very erotic; after her marriage at that age she became frigid. Three months after her marriage, following a mental strain, there came the first indications of the disease, in the form of weakness and paresthesia in the hands. The sensation of touch remained for a long time after discarding an object. When she had dropped a key to the floor, she seemed still to feel it in her fingers; even after voluntarily laying aside a thing she would have in her hand the persistent sense of still touching the object she had put aside. In

walking the soles of her feet felt as though they were treading upon a soft, yielding surface, like a pillow; the gait was unsteady and reeling, and standing was difficult. There were no pains; voice and swallowing were normal. After a gradual increase of symptoms for four weeks, followed by six weeks' treatment by electricity at the London Hospital, she recovered sufficiently to return home.

Immediately upon her return she found that the house had been robbed, and the shock caused a return of the previous symptoms, together with diplopia and flaccidity of the legs. At the National Hospital, where she was treated during this attack, Dr. Bastian was said to have diagnosed the case as weakness in the legs, due to worry. After this attack she made apparently a complete recovery, and was entirely well for years.

Ten years ago, after the death of her father, there was a third attack, with a return of the weakness in the legs and unsteady gait, which lasted two months before the patient recovered.

Her recent exacerbation came on a year ago, and during this time the former symptoms returned with steadily increasing force. The gait again grew difficult and unsteady, the lower extremities weak, with a twitching of muscles of the leg, and a stiff feeling below the knees. There was imperative urination. The singing voice was lost. During this, as well as the other attacks, there was no loss of emotional control.

Her status, on examination at Mt. Sinai Hospital, November 22, 1914, presented unmistakable characteristics of multiple sclerosis.

Eyes: Small; pupils have a sluggish light reaction; nystagmoid movements in extreme positions; bitemporal pallor of the discs. Reflexes: Oppenheim; Romberg; bilateral Babinski present; absent abdominal. Tendon: U. E. diminished, lower present. Motor control: Spastic face; ataxic gait; hypotonia; slight unsteadiness in left hand at times; marked unsteadiness in the legs. Heart: Systolic apical murmur. Viscera: normal. Wassermann and spinal fluid: negative. Also a case of multiple sclerosis.

ETHICAL ASPECTS OF PSYCHOANALYSIS

By John T. MacCurdy, M.D.

(By invitation)

Psychoanalysis is a method of psychological investigation and therapy, which with its theory stands in a peculiar position to-day, inasmuch as both its opponents and its adherents show a spirit in their attacks or defense, so strong as really to be inconsistent with dispassionate scientific argument. There must be something in it, therefore, which stirs an emotional response. What this is may be inferred from the fact that the ranks of psychoanalysts have recently been split on that very rock which is the target for the abuse of the opponents. This is the claim that unconscious sexual motives, particularly of abnormal cravings, are basic dynamic mental factors. The claim that a mental cause may produce disease meets naturally with opposition from those who are convinced that there must be a physical abnormality in the brain corresponding specifically with every mental symptom. But equally bitter attacks come from those who are open to a belief in psychogenesis. The crux of the situation is therefore in the nature of psychoanalytic claims.

It is necessary to examine these claims from two standpoints—that of objective validity, and that of their ethical value or potentiality for good and evil. The evidence in favor of the truth of the teachings of psychoanalysis rested originally on the method of free-association. As this method implies a familiarity on the part of the physician with his technic such as is only gained by experience, and as the patient to coöperate must have established a certain *rapprochement* with the analyzer, it is difficult to demonstrate *objectively* that no suggestion is at work. It has been found, however, that the history of myths and folk-lore shows a development of ideas identical with those which Freud claimed to find in the unconscious of the neurotic individual. This would seem to offer strong support of his theory. But more recent work with the psychoses has shown that there is no "complex" reported by Freud which does not reach open expression in the psychoses under conditions which preclude suggestion. Moreover, the relation of these normally unconscious ideas is so regular that it is now often possible to predict the delusions or hallucinations to arise in a psychosis, knowing once the make-up of the patient and the precipitating cause of his break-down, although there may seem to be no surface relation between these factors whatever. This is such a flawless objective proof, as it reduced the problem to experimental conditions, that no psychiatrist can now be scientifically honest and deny the substance of these claims.

It is important, however, to examine the ethical aspects of psychoanalysis, for if the matter concerned be morally repulsive, anyone may be pardoned for paying no attention to it. That psychoanalytic claims are often regarded as being repulsive is certain. How has this come about? It is probably owing to two misconceptions: that what is unconscious is to be judged by the same standards as what is conscious; and that a symbolic outlet to a forbidden wish is as reprehensible as a direct fulfilment of such a wish. It is, however, obvious that if any idea be unconscious it can have no relation to reality. If not in contact with reality it cannot lead to any overt act. Immorality implied an act or at least the thought of some act; but an unconscious idea cannot be spoken of as immoral or even dangerous, so long as it remains in the unconscious. If the history of these wishes be followed, they are found never to have been in contact with reality, never fully conscious. This may be seen in the growth of the unconscious wish for incestuous outlet with a parent. The child loves his parent and naturally strives for expression of his affection. As soon as sex consciousness begins to develop, it is inevitable that the idea of physical outlet with those he most loves should appear. In so far as this outlet is frankly sexual, however, it is repressed into the unconscious. That the wish exists in the unconscious only is therefore a tribute to the morality of the individual. When these wishes do get into consciousness, it is only by symbolic expression. These symbols are symptoms, or sublimations. In both cases there is a distortion which makes the final concept perfectly acceptable to the moral side of the subject, except in such unusual cases as the kleptomaniacs. Sublimations are more than mere distortions; they are outlets which are socially useful. The individual in this case then uses the energy of his forbidden wish to benefit mankind. In demonstrating this, psychoanalysis pays a tribute to the morality of mankind rather than debases it. Both repression and sublimation demonstrate an ethical power in human beings, previously unguessed.

Dr. August Hoch said that he thought all must have been impressed

with the atmosphere created by this paper of Dr. MacCurdy's, an atmosphere different from that which the opponents of psychoanalysis have been accustomed to associating with the subject, for to them it was a subject in which nothing else but sexuality was dealt with, more particularly sexuality as they conceived it. Such an exposition of psychoanalysis as Dr. MacCurdy had just given should go far toward correcting such an impression. The psychiatrist had of course long been impressed with the frequency with which sexual motives were found in the psychoses, and therefore it was not surprising that a similar state of affairs should be found in the closely related neuroses. He thought that the most fundamental part of Freud's teachings, namely, that of the importance of the imperfect development of the love instinct, was proved by studies in the field of neuroses and psychoses; but he felt that Dr. MacCurdy had done a distinct service toward a better understanding of psychoanalysis by laying stress, not only on the more individualistic side of our instinctive life, but on the social side as well, *i. e.*, the moral, adaptive forces which represented not only the ethical aspects of psychoanalysis but of the neuroses and psychoses as well.

Dr. Steinach asked Dr. MacCurdy how he could explain the Abderhalden reaction which recent investigators have shown to be fairly constant in dementia præcox, if the disease were of purely psychogenetic origin as he seemed to indicate in his paper.

Dementia præcox seemed to the speaker more like a chronic intoxication due to perverted metabolism and probably resulting from disarrangement of some of the internal glandular secretions.

With equal propriety one might make a psychoanalysis of any intoxication such as alcoholism or the delirium of typhoid fever, but the conclusions arrived at would in no respect help to solve the question of the origin of the condition.

Dr. Clark said apropos of critical remarks made concerning the much vexed question of the pathogenesis of dementia præcox, while it must be recognized that in certain mental diseases there are metabolic changes, yet this fact does not necessarily make observations of its manifestations of no value in the effort to gain some clue to its primary causes. Allowing the position of Dr. Steinach its full force, what had he to say with reference to the undeniable similarity of mental trends in organic nervous diseases and those psychopathic conditions where there is no manifest organic change? Where like physical causes were not operating to produce like effects, certainly it seemed reasonable to explain similar trends as due to some related underlying cause of a different nature, which the Freudian theory does by attributing both types of mental disorders to defective adaptation of primary instinctive trends to adult purposes in life.

Dr. Smith Ely Jelliffe said that he had nothing to add to Dr. MacCurdy's paper, except that it seemed to him to set forth what might be called the trend of modern opinion regarding psychopathic cases and that it was of value to have had a presentation of the subject which, as Dr. Clark and Dr. Hoch had just said, should go far toward creating in the minds of those hearing it a clearer conception of certain commonly misunderstood phases of the subject.

Freud explains very distinctly that neuropathic disorders have their rise in the reluctance of the subject to recognize certain vital situations which offend the moral side of his nature; rather than consider the possi-

bility of taking any step downward in his ethical attitude toward certain social reactions, the patient is ready to suffer martyrdom.

To brush aside an explanation of such conditions by such a sweeping objection as, "But these are abnormalities," reminded the speaker that in a celebrated dialogue of Protagoras a similar attitude was most strikingly disposed of. It might bear repetition at this late date since one hears it repeated so frequently in discussions of a so-called logical type. Here Protagoras called the attention of Morosophus, his adversary, to "Xenias the son of Glaucus," asking Morosophus if he knew him.

Morosophus: Yes, but he seemed to me a very ordinary man and quite unfit to aid in such inquiries.

Protagoras: To me he seemed quite wonderful and a great proof of the truth I have maintained. For the wretch was actually unable to distinguish red from green, the color of the grass from that of blood! You may imagine how he dressed, and how his taste was derided. But it was his eye, and not his taste, that was at fault. I questioned him closely and am sure he could not help it. He simply saw colors differently. How and why I was not able to make out. But it was from his case and others like it, but less startling, that I learned that truth and reality are to each man what appears to him. For the differences, I am sure, exist, even though they are not noticed unless they are very great and inconvenient.

Morosophus: But surely Xanthias was diseased, and his judgments about colors are of no more importance than those of a madman.

Protagoras: You do not get rid of the difficulty by calling it madness and disease. And how would you define the essential nature of madness and disease?

Morosophus: I am sure I do not know. You should ask Asklepios.

To which Protagoras remarks: "Ah! he is one of those gods I have never been able to meet!"

Dr. MacCurdy, after expressing his appreciation of the cordial reception which his paper had met, said the fact that it had not given rise to more discussion by those holding contrary beliefs was to him a disappointment, since he felt sure that a talk extending over some fifteen minutes' time could scarcely have done a great deal toward changing views; he regretted that there had not been more adverse discussion of his paper, particularly since he had grouped the points made therein in such a fashion as to make it possible to take them up one at the time, with the idea of making them the starting point rather than the close of discussion.

Referring to Dr. Steinach's question, Dr. MacCurdy said that he presumed Dr. Steinach used the Abderhalden reaction as a specific example of a toxic influence which might be the causative factor in dementia præcox. That organic factors were important in dementia præcox is not only admitted by psychoanalysts, he claimed, but psychoanalysis furnishes a most definite proof of the existence of such factors. If normal, neurotic and psychotic individuals all had identical unconscious "complexes," then the difference these different types of individuals show in meeting certain situations common to them all could only be explained by the dementia præcox individuals' having from the outset an inelasticity and a defective adaptation, which must be founded on a physical basis. Any more specific statement of the relation between insane ideas and diseased cortex was impossible. The origin of definite ideas could only be traced by psychological means; a physical correlation implied a knowledge of what the

mechanical or chemical properties of the ideas were. That, of course, was ridiculous in the light of our present ignorance of such things.

SOME OBSERVATIONS ON DEMENTIA PRÆCOX

By Walter L. Treadway, M.D.

(By invitation)

The paper dealt with dementia præcox in men, and called attention to the fact that it is no longer to be questioned that in the genesis of dementia præcox the so-called shut-in type of personality is essentially the type that breaks down with this disorder. The original description of the shut-in type of personality left out, to quite a considerable extent, the abnormalities in the sexual life of these individuals. Dr. Treadway pointed out that in the development of sexuality certain types of shut-in personality show an inability to fuse the tender feelings and the sensual feelings, and to bestow the two together on an adult object. The Oedipus situation plays the important part in preventing the biological demands of adult life properly to adapt itself,—i. e., an inability to replace the infantile tendencies with an adult object,—hence a defective adaptation in love affairs, mating, and propagation; in other words, a defect in the instincts.

The first case considered was that of a normal man with externally an excellent adjustment to life, but with evident difficulties in the sexual sphere. He was quite capable of having sexual relations with women, but for them he had no tender feelings. He was also quite unable to have any erotic desire for a woman for whom he had a real feeling of love or reverence. Two women for whom he held this tender feeling were older than he, and of them he drew a plain parallel with his mother, so that both represented mother substitutes. This case was presented with a view to bringing about a better understanding of the cases which followed.

The article was not confined to the make-up of dementia præcox cases, but to some points about the precipitating causes, and about the content of the psychosis.

The first case of dementia præcox presented a shut-in personality with the following abnormal traits in his sexual life.

Although the purely sensual side of his sexuality seemed not to be repressed, he was decidedly defective in his capacity to make love and to contemplate marriage seriously. Such abnormalities were considered an evidence of an infantile attitude, so far as love and marriage were concerned, and represented an inability to develop beyond the mother attachment. This case showed that the man's ideas which referred to his getting an independent position in life, and the finishing of his apprenticeship, were closely related with the idea of marriage, and owing to his make-up it was precisely that from which he shrank so much. It was then regarded that the taking up of a definite life work, and the final coming into sight of the termination of his apprenticeship, represented the precipitating cause, or the precipitating cause acted in the same direction in which the defect of his make-up tended. The content of his psychosis was marked by homosexual features, but there was also another part which represented the wish not to go through the apprenticeship, not to have sexual power, all of which was connected with his ideas of marriage

The trend was an expression of a regressive sexuality; that is, of non-adapted love; namely, in this case homosexuality. So that make-up, precipitating cause, and content were the expression of the same innate tendency or defective adaptation.

Two other cases were briefly reviewed.

The second case of dementia præcox was that of a typical dementia præcox make-up. But in this case the subject broke down, not under the demand for adult love, but when his father died. A manic-depressive personality would have developed a retarded depression, but in this case the subject developed a crude infantile sexual trend. At first glance it looked as if the principle of the two cases were very different, but much similarity was in evidence. In the first case of dementia præcox the patient broke down because he was unable to meet the demands of adult love; he was unable to do that on account of his infantile tendencies. In the second case of dementia præcox no demand was made, but the infantile tendencies became suddenly inflated through the death of the father, *i. e.*, his rival. The patient presented a very infantile trend, a cosmic formulation, of having relations with his mother, of being his father, and of recreating himself with his mother. The principle, however, that the make-up, the precipitating cause, and the content of the psychosis were closely connected and worked all in the same direction, was again evidenced in this case as well.

The third case of dementia præcox showed a very marked incapacity for adult love which stood distinctly in the foreground, and was rather more marked than his general shut-in tendencies. No definite precipitating cause was demonstrated. But the case showed an interesting feature which formed a parallel to the precipitating causes, especially that of the second case of dementia præcox reported. He changed his habits after the father's death, began to wish to appear more manlike, drank, and became an agitator. The inflation which the unconscious tendencies received after the death of the rival was in this case not transformed into psychotic symptoms so much as into fairly well adapted habits,—in other words, sublimated. He got an outlet in more association with men, in drinking with them, and in becoming a revolutionary spirit. His trend was essentially a homosexual one. Aside from the usual homosexual accusations, his ideas chiefly were that he was changing into a woman. Here, as in the first præcox case, one of the typical escapes from real love destiny was chosen, the path of unreal adult love, that of homosexuality. In this case the trend and the make-up were the result of the same tendencies.

The following points were brought out:

First: That the adaptation defect in the sexual sphere, although it may express itself in a complete shunning of all sexual relationship with the opposite sex, may often not lead to any marked repression of mere sensual sexuality, but may gravely interfere with the capacity to meet the demands of adult love,—*i. e.*, marriage.

This is of course a trait not confined to dementia præcox, but is one of the ways in which, as Freud has shown, the defect of sexual adaptation may manifest itself in any neurotic individual. The first case reported was a good example of this.

Second: That when we fully appreciate the internal unconscious meaning of the defect of sexual adaptation,—namely, the fixation in an infantile state of attachment to the parent of the opposite sex—it is clear that the

peculiarities of make-up, the nature of the precipitating causes, and finally the content of the psychosis are very closely related, in that they all are dominated by the incapacity to fulfil the demands of adult love, and the desire to get away from this.

The trends described in the cases here reported are either a direct expression of the desire to avoid marriage, or they represent an escape in the sense of homosexuality, or they are directly of the nature of plain infantile sexuality.

Dr. Hoch stated that it seemed to him very valuable to bring out the points which Dr. Treadway had just developed, by demonstrating the possibility of viewing the make-up, the precipitating cause, and the ideas expressed by the patient (the so-called content of the psychosis) from a common point of view, namely, from that of an underlying defective development of instincts. He stated that after the earlier studies on make-up in dementia præcox, which had led more to a surface description of the essential traits of the constitutional abnormality, it had more recently become possible to demonstrate that the stunted development of the adult love instinct represented an important component of the make-up of dementia præcox cases, and that this inadequate development also showed itself in the ideas expressed by the patient, which betrayed motives belonging to an earlier stage of development.

Seeing a significant point in connection with one of the cases presented by Dr. Treadway, that of the workman who as soon as he had finished his apprenticeship found it impossible to go on doing his work, Dr. MacCurdy said that a striking thing in this instance was that no difficulty whatever had been experienced with the work until a time was reached when the training was finished and the man had reached an independent position; everything went smoothly until that particular period was reached which, according to the confession of the patient, was identified with the assuming of a certain responsibility,—that is he was now a worker for a higher wage and could afford to marry. Confronted by the removal of all obstacles that heretofore had deterred him from this step, which he seemed to consider a duty, he suddenly found it impossible to do the same sort of work which he had been doing before to the satisfaction of those who employed him. Evidently, at the time of the breakdown, the work was no longer viewed as a thing in itself, but rather was unconsciously considered in the light of its associations. A knife which a man carried around with him, using it to cut his bread, or sharpen a pencil, is of course to be regarded as simply a knife. But when a man feels that the knife *must* be carried, through the force of some compulsion he would be at a loss to explain, which fills him with a feeling of gone-ness if he does not have it with him, then, to him, the knife has ceased to be a mere knife, and has assumed an importance that can be explained only as being symbolic. So, in the case under discussion, it seems reasonable to conclude that the illness came not from any strain directly attributable to the work itself, but rather from conditions which in the patient's mind had become identified with the finish of his apprenticeship and the obtaining of an independent position in life—conditions which, since he felt it impossible to meet them, put in motion the associated feeling of an inability to go on working, as he heretofore had done.

Dr. Clark commented upon one of the cases presented in Dr. Treadway's paper, that if it be true that the primary fault in dementia præcox is an inherent inability in the individual to adapt himself to the socialistic tendencies of adult life, and particularly to the main purposes of a proper

marriage, then is it not possible for us to see some therapeutic advantage in this conception? If one may be fairly certain of this position, why not arrange the lives of the *præcox* to the simpler adolescent tasks whose trends do not lead essentially into situations that call for these impossible demands? Industrial occupations, shop, farm and garden work and life led apart from the temptations for heterosexual life, may conserve the *præcox* from the deterioration consequent from being measured up to a standard he cannot meet. This position would possibly render stationary the cases which are first made quiescent by hospital treatment; but to be fully effective the *præcox* would have to be diagnosed and placed under treatment early, whereas at present only too frequently he is not even seen by physicians. The early detection of the sinister import of the shut-in type of personality in adolescence is very imperative as we gain a truer insight into the essential pathogenesis of the disorder. All agencies dealing with children should be keenly aware of some of these newer studies in order that the above conservation principle of treatment may be applied.

Translations

VAGOTONIA

A CLINICAL STUDY

BY PRIVATDOZENT DR. HANS EPPINGER AND DR. LEO HESS

OF VIENNA

TRANSLATED BY WILLIAM MAX KRAUS, A.B., M.D., AND
SMITH ELY JELLIFFE, M.D., PH.D.

(Continued from page 250)

We shall now try to present some basis for this idea:

We have frequently observed that vagotonia occurs most often in young people. Experimental pathology also has taught us that a higher vagal tone is to be expected in young animals than in adults. Furthermore, according to Bartel.³⁵ Lymphatism is found with decreasing frequency as age increases. Since we know that the thymus disappears after a certain age, while in youth it is almost a physiological organ, the above-mentioned facts cannot fail to be both striking and noteworthy. We believe also that we can derive some additional proof from Graves' disease, which is so often associated with a persistent thymus. We have learned from the researches of Tacher that in Graves' disease an accumulation of lymphoid tissue occurs in the thyroid and in the more usual places, where this type of cell is found. Further that this is more pronounced the longer the duration of the disease has been. This suggested the hypothesis that a substitution or healing process was taking place, the lymph tissue growing to replace some diminution in the activities of some other organ. This idea had been advanced by earlier authors. Mikulicz recommended thymus preparations in the treatment of Basedow's disease, and his reports leave the impression that some good was

³⁵ Bartel, J., Über Konstitution und Krankheit. Verhandlungen der Deutschen pathologischen Gesellschaft, 1910, 14 Tagung.

done by its administration. We ourselves have noted that lymphatism and the signs of persistent thymus were most pronounced in the markedly vagotonic forms of Basedow's disease. And it is of further interest that our vagotonic showed an eosinophilia and relative lymphocytosis, the same as in the vagotonic Basedow cases. Recently Bartel ⁸⁶ has studied the question of status thymicolymphaticus from the anatomic point of view.

He concludes that lymphatism and persistent thymus are part of a general state of inferiority, one of whose manifestations is a decreased resistance to many noxious influences. What interests us most, however, is the coincidence of lymphatism with just that group of diseases which we ourselves have found associated with vagotonia. He finds, for example, that certain diseases are never associated with a lymphatism, while in others it is a frequent finding. It is of special significance that Bartel never found carcinoma and lymphatism associated, while cases with sarcoma, a disease of youth, frequently showed lymphatism as well.

He proposes the theory that there exists in the constitutional inferiority of status thymicolymphaticus some factor which predisposes to certain diseases and against others. Now the fact is that just those diseases which we have found to be vagotonic in nature, or which are readily developed upon a vagotonic basis, are, according to Bartel's findings, combined with lymphatism. This may be shown in part by clinical observations. We have shown that in many cases of vagotonics there is enlargement of those parts of the lymphatic system which are enlarged in status thymicolymphaticus. More accurate proof we have not, since this proof is based on anatomical findings. Probably the anatomical entity status thymicolymphaticus corresponds most closely to the clinical entity, exudative diathesis, which Czerny has described. But there is great difficulty in determining this clinical entity anatomically, since in children it is difficult to set a limit between normal and enlarged lymph tissue. Clinically Czerny's exudative diathesis corresponds most closely to vagotonia. One may even go so far as to say that the exudative diathesis, with its tendency to spastic states and convulsions, to vascular disturbances and exudations, is but an infantile form of

⁸⁶Bartel, J., "Über die hypoplastische Constitution und ihre Bedeutung," Wiener klin. Woch., 1908, No. 22.

vagotonia. It is greatly to be desired that pediatricians should aid us by an analysis of the symptoms found in children with exudative diathesis. Our theory is much strengthened by Strümpell's observation that those who are affected with bronchial asthma are, perhaps, of the class who have had the exudative diathesis in childhood.

It may be seen that we have found in our conception of vagotonia a way to discover in various types of individuals a definite constitutional state, which in some cases may predispose to disease, in others may give a disease a definite course. This has given a degree of unity to phenomena which hitherto have been considered both clinically and anatomically as due to various kinds of predisposition.

The criterion, which clinicians and anatomists have used to describe the origin of these predispositions, has up to now been only morphological. The important thing has been the lymphatic tissues, and their reaction to various stimuli. Nothing could be said of the function of this reaction, except that it was associated with some degree of constitutional inferiority.

Since statistical studies have shown that length of life as well as morbidity are closely related to these morphological changes, one may say that lymphatism, combined with status thymicus, comprise a remarkable constitutional state of inferiority, and is related to a predisposition to certain definite diseases.

Our attempt to determine a form of predisposition by functional tests of a chemical nature brings up the very interesting question, is there any parallelism between a clinical and anatomic predisposition? In our opinion this parallelism may be doubly demonstrated. Just as Bartel states that lymphatism is a partial manifestation of constitutional inferiority, and is frequently associated with developmental anomalies, so do we believe that vagotonia is a partial manifestation of an inferior organism. We feel that the chromaffin organs, like other organs, may be arrested in their development. Thus adrenalin would be produced in lesser amounts, and its antagonist would take the upper hand. This should be considered particularly in cases of general vagotonia.

There are many clinical proofs that defective development of the adrenals, and thus their functional activity, plays an important part. We find that cases of vagotonia have enteroptosis, are of

the degenerate type of individual, and frequently have neurasthenia, hysteria, high arched palates, flat foot and syndactily in association with the symptoms of the vagotonia.

We must also consider the possibility that vagotonia, which improves with advancing age, and in fact may even disappear, and yet, under certain pathological conditions may reappear, may be closely related to an overactivity of some of the glands of internal secretion. Thus a state would be produced by this change in the interrelations of the glands of internal secretion, which could certainly not be considered an anatomic form of constitutional inferiority.

We feel that vagotonia may very probably be the clinical component of the anatomical lymphatic constitution.

The parallelism between the clinical picture of status thymicolymphaticus, uncertain though it may be, and the readily demonstrable anomalies of vagotonia, makes us very prone to believe our theory of their relation to be true. The fact that we could not give a clinical demonstration of status thymicolymphaticus in many of our vagotonic cases, is not weighty evidence against our theory, since status thymicolymphaticus may only be demonstrated anatomically. Furthermore, it is unusual to have a typical vagotonic come to autopsy, since vagotonia occurs most usually in the youthful. Yet we were able in a few cases to confirm the parallelism of vagotonia and status thymicolymphaticus at autopsy. We hope to be able to demonstrate this experimentally.

In closing, we wish to say a word about the etiology of the mixed forms of vagotonia. Perhaps these are due to a polyglandular disturbance of endocrinous nature. Thus the picture of vagotonia would be obscured. Just as the typical action of physostygmmin is obscured by using curare with it. At all events, these cases offer a very fertile field for clinical study.

(To be continued)

Periscope

Review of Neurology and Psychology

(Vol. XII, No. 6)

1. The Nature and the Treatment of the So-called "Genuine Epilepsy,"
G. C. BOLTEN.

2. Notes on a Case of Pellagra. D. MAXWELL ROSS.

3. Babinski's "Second Sign" of Organic Hemiplegia in Hemichorea, and its Bearing on the Organic Nature of Chorea. ARTHUR F. HERTZ.

1. *Treatment of So-called "Genuine Epilepsy."*—A summary of this article is as follows:

1. Of the numerous cases of epilepsy which make their appearance quite as genuine epilepsy, an important part, depending upon collective, divergent, primary cerebral affections, belongs to the cerebral (secondary, symptomatic) epilepsy, and but the smaller part (± 25 per cent. concerning our cases) to the so-called genuine (essential, idiopathic, or primary) epilepsy.

2. Even now it is quite impossible to distinguish, less on pure clinical symptoms, most cases of cerebral from genuine epilepsy.

The differential diagnosis between these divergent affections must move itself in the sphere of the pathology of metabolism.

3. Genuine epilepsy is a chronic auto-intoxication, caused by a hypofermentation of the intestinal tract and of the intermediary metabolism, as a consequence of a hypofunction of the thyroid and parathyroid glands (or of troubles in their nervous elements, to know the sympathetic nerve).

4. Genuine epilepsy is quite curable by the administration, rectally, of fresh pressed extracts of the insufficient glands, or better, it is possible, in this manner, to free the patient from all symptoms, as I have proved in a great series of cases.

2. *Notes on a Case of Pellagra.*—The patient was a mill girl from Leith and was treated at the Royal Edinburgh Mental Hospital. Mentally, she was at times mildly delirious, and when not delirious was depressed and neurasthenic. The alimentary symptoms were diarrhea with hemorrhoids. There were no gastric symptoms. No stomatitis is noted. Neurologically, there was the usual exaggeration of the reflexes; and death occurred in clonic convulsions. The dermatological symptoms were very slight and transient in character, but were the first to attract attention. They were described by the nurse in charge as a "bad case of sun-burn." The hands were the chief site of the lesions. The lesions were characteristic. Bacilli pyocyanus were found in the intestine. On post mortem the calvarium and the meninges were found much injected, and in several places on the cortex there were minute pial hemorrhages. There was marked excess of cerebro-spinal fluid. The brain was soft, and weighed only $43\frac{1}{2}$ ounces. Subsequent microscopic examination of tissue removed from the cortex showed little pathological change. There were no definite degenerative changes, no arteritis, and no increase of neuroglia. Unfortunately, the cord was not removed.

3. *Babinski's "Second Sign" in its bearing on Chorea.*—This sign was described by Babinski as "combined movement of the trunk and

pelvis." In organic hemiplegia, in attempts to sit up from the dorsicumbent position, the paralyzed leg always rises higher than the other. In seven (7) cases of hemichorea, tested by the writer, the leg on the affected side rose higher than the other although the plantar reflexes were both flexor and the cutaneous and tendon reflexes were equal on the two sides. As this sign has never been observed in any case of hemiplegia which was not organic, its constant presence in hemichorea points strongly to the conclusion that the upper motor neurone is affected by some organic change in hemichorea, even if it is of a kind which escapes recognition by the ordinary methods of histological examination, and as Babinski's second sign is constantly present in hemichorea, it may reasonably be concluded that the lesion with which it is associated is constantly present in ordinary cases of bilateral chorea. As this sign is the only evidence of the existence of an organic lesion in hemichorea, and as it depends on a comparison between the two sides, direct evidence of the organic nature of the nervous symptoms in ordinary chorea is still wanting.

C. E. ATWOOD (New York).

Journal of Mental Science

(Vol. 57, No. 238)

1. Morison Lectures; The Differentiation of Melancholia, the Depression of Manic-depressive Insanity. GEORGE M. ROBERTSON.
2. The Pathogenesis of a Delusion. HENRY DEVINE.
3. Witchcraft, Demoniactal Possession, and Insanity. HUBERT J. NORMAN.
4. Some Instances of Sudden Death with Post Mortem Findings. J. FRANCIS DIXON.
5. Asylum Dysentery. W. J. ADAMS ERSKINE.
6. Syphilis and Congenital Mental Defect. C. G. A. CHISLETT.
7. The Treatment of Puerperal Insanity with Anti-streptococcic Serum. NATHAN RAW.

1. *Differentiation of Melancholia. Morison Lecture.*—A complete discussion of melancholia or the depressive phase of manic-depressive insanity, with which classification the author is in accord. The classical symptoms such as depressed or painful emotions, retardation both mental and motor, delusions explanatory in character, tendency to suicide, et cetera, are described in detail. The importance of a thorough physical examination and the careful treatment of any disorder, the necessity for the supervision of the feeding of such cases to guard against starvation, and the need for constant watchfulness to prevent suicide, are all emphasized.

2. *Pathogenesis of a Delusion.*—Devine calls attention to the importance of going beyond the formal classification and prognostic determination in examining an individual case. In illustration of the value of a closer study, he has, by association test and subsequent analysis, traced the development of a hypochondriacal delusion with reëducation of the patient and final recovery.

3. *Witchcraft and Insanity.*—In a general discussion with historical examples, the writer points out that primitive religion was based on the long existing and universal belief in evil spirits. It was but a short step to witchcraft, and misapplication of Scriptural texts, with hysterical or insane actions and utterances wrongly interpreted, coupled with superstition and ignorance, gave rise to centuries of persecution.

4. *Sudden Death with Post Mortem Findings.*—Dixon narrates cases in which sudden death was an unexpected termination. Among these were (as determined by autopsy) septic infarcts of the lung from a sacral bed-sore, tumors and abscesses of the brain, aneurysms of various localities and obliteration of a coronary artery by endarteritis.

5. *Asylum Dysentery.*—After enumerating the recognized etiological factors of dysentery, i. e., overcrowding, possibly constipation, dirty habits of the insane, peculiar condition of the nervous system, and the presence of dysentery carriers, Erskine discusses three other causes. He is of the opinion that the presence of kidney disease, tuberculosis or influenza renders cases more liable to dysentery. In the case of influenza, that alone may be a potent cause for the intestinal condition as was shown in an epidemic described.

6. *Syphilis and Congenital Mental Defect.*—Chislett reports the results of Wassermann reaction in a number of cases of congenital mental defect. Out of fourteen idiots and imbeciles eight gave a positive Wassermann. Three cases of juvenile general paresis gave positive reactions. Out of three epileptic idiots, only one gave a positive Wassermann. Three paralytic idiots gave negative reactions. A whole family in which the father was an acknowledged syphilitic was examined. There were ten pregnancies, two children premature, and two died in infancy. Of the six living children, three gave positive reactions, only one of which showed suspicious clinical signs, although one other was nervous and stupid. The mother had a tertiary lesion on the leg eight years after marriage.

The writer calls attention to the possibility for prophylactic measures to prevent congenital mental defect.

7. *Treatment of Puerperal Insanity.*—Raw states his own conclusions as follows: 1. Puerperal insanity is only rarely if ever caused by septic infection at the puerperal period. 2. Anti-streptococcic serum of a polyvalent nature seems to exert a very favorable influence in many cases of puerperal infection, but does not appear to influence the course of the mental process. 3. Puerperal insanity, of whatever variety, is most favorable for cure in a properly equipped mental hospital, provided that the treatment is undertaken immediately after its onset.

The question as to what constitutes so-called "puerperal insanity" is not discussed.

W. C. SANDY (Kings Park, N. Y.).

(Vol. 57, No. 239)

1. Presidential Address. Relation between the Geographical Distribution of Insanity and that of Certain Social and other Conditions in Ireland. W. R. DAWSON.
2. The Personal Equation in Alienism. THOMAS DRAPES.
3. Psychotherapy in Mental Disorders. WILLIAM GRAHAM.
4. Note on Hereditary Insanity from a Practical Standpoint. R. R. LEEFER.
5. Causes of Sudden Death in Epilepsy, and Some Points in the Treatment of Epilepsy. M. A. COLLINS.
6. Mental Symptoms in Association with Choreiform Disorders. EDWARD MAPOTHER.
7. A Report on the Bacteriological Investigation of the Blood in Fifty Cases of Insanity. W. T. SEWELL and COLIN McDOWALL.
8. On Mental Inspection in Schools. SIDNEY J. STEWARD.

9. The Deviation of Complement in Cases of So-called Idiopathic Epilepsy. G. H. GARNETT.

1. *Geographical Distribution of Insanity and Other Conditions in Ireland.*—From a study of statistics, Dawson reaches the following conclusions. In Ireland, insanity tends to prevail in the agricultural counties and has a close relation—if special conditions be disregarded—to pauperism, which also prevails in the rural districts. The distribution of insanity corresponds to but a very small extent with the emigration rate. It bears some little relation to the prevalence of criminality, and to that of chronic alcoholism. No appreciable relation is apparent between the insanity rate and density of population, rateable valuation of land, distribution of the number of aged persons, death rate (either general or tubercular) or drunkenness. The amount of insanity ascribed to alcohol is small and its distribution has no relation to that of drunkenness but some to insanity in general. Pauperism in Ireland bears little relation to poverty and does not correspond to the rateable valuation of the land nor is it closely connected with emigration. Criminality, as in England and Wales, is greatly in excess in the large towns, the rural districts being relatively free; but on the other hand, in Ireland drunkenness is found to be more prevalent in the rural counties which is the reverse of the condition across the Channel. A number of tables are appended upon which the above conclusions are based.

2. *Personal Equation in Alienism.*—Drapes calls attention to the opportunity that exists for varied conclusions in the estimation of a given set of phenomena in the different fields of science such as psychology, physical science, etc. In alienism, it is especially seen in classifications and he mentions the dementia præcox controversy that exists in England as an illustration. Moreover, there is a difference in the reaction of different individuals, races and so forth, to the same disturbing agency, making another element to be considered in each individual case. The author mentions a former paper in which he advocated the idea of the unity of insanity, one malady throughout, but manifesting itself in many forms and phases, but not to be regarded as distinct disease entities.

3. *Psychotherapy in Mental Disorders.*—After calling attention to the fact that the English have taken little interest in psychical methods of treatment as compared with the French, Germans and Americans, Graham discusses the question under these heads: (a) Suggestion, waking and hypnotic, (b) therapeutic conversation, (c) psychoanalysis, (d) occupation, (e) reëducation. In the general discussion following, widely different opinions as to the efficacy of psychotherapy are expressed.

4. *Note on Hereditary Psychoses.*—Leeper calls attention to the defects of a Poor Law which allows reproduction to go on among the feeble-minded both legitimately and illegitimately, a constant source of hereditary insanity. He quotes Burton's "Anatomy of Melancholy," where it is said that in Scotland men with certain dangerous diseases which are likely to be propagated "are gelded," women kept from the company of men, and so forth, showing that the principles of eugenics were apparently well recognized even in the 17th century.

5. *Causes of Sudden Death in Epilepsy.*—Collins investigated ten cases, and found the cause of death in two was choking from particles of food, in one suffocation during fit, in one mitral and aortic disease, adherent pericardium in two, advanced disease of the heart (fatty) in three, and cerebral tumor in one. In only one case was the brain congested. This is in sharp contrast to the findings of Marchand who, out of fifteen cases,

finds five of ruptured heart or aorta, and six by rupture of cerebral or meningeal vessels. Twice was congestion of the brain noted as the sole finding. The author also discusses treatment of confirmed epilepsy, favoring colony life, a reasonable diet and interesting employment in the open air, with bromides to control excess of fits, malaise or excitement.

6. *Mental Symptoms in Choreiform Diseases.*—Mapother names the conditions in which choreiform movements may appear as follows: (1) Sydenham's chorea or chorea minor, (2) Huntington's chorea, (3) chorea dependent upon gross organic brain lesion, (4) "senile chorea," (5) hysterical chorea. Associated with Sydenham's chorea, there may be severe delirium-like symptoms or the symptoms of manic-depressive insanity, manic phase. Such cases occur commonly during pregnancy. There are also cases in which marked dulness, apathy or despondency characterizes the mental state. In Huntington's chorea, there is a gradual progressive mental deterioration extending over years, with exacerbations. During the latter, there may be impulsive acts of irritability, hallucinations and delusions. The mental symptoms associated with chorea dependent upon gross organic brain lesions are not characteristic but depend upon the location of the lesion. Senile chorea in some cases resembles Huntington's chorea, in other cases seems to be the result of diffuse arteriosclerosis. Associated with hysteria, chorea may be either rhythmic or arrhythmic. The former is most common and occurred in epidemics during the fourteenth and fifteenth centuries under the influence of religious excitement. The movements are accompanied by amnesia and may be checked by suggestion. Arrhythmic cases are commonly imitations of some case of Sydenham's chorea seen by the patient. Interesting features are the sudden onset, and prolonged course, also the readiness with which it may be cured by suggestion.

7. *Bacteriological Investigation of Blood.*—Sewell and McDowell investigated twelve cases of excitement with confusion, 27 of melancholia, and eleven of chronic psychoses with the hope or expectation of finding microorganisms in the blood. In only one case were any found and in this it was unimportant so far as the mental condition was concerned. In one case *Staphylococcus pyogenes aureus* was found, but this patient had attempted suicide by gashing her throat, and the infected wound accounted for the microorganism.

8. *Mental Inspection in Schools.*—Steward, a school inspector, recommends that all backward or abnormal school children be examined around the age of nine to eliminate them from the ordinary school course and furnish them with special tuition. He outlines a printed form for the examination, consisting of first, a teacher's confidential report on the standard of school work, character, habits etc.; second, psychological examination, with anamnesis, physical and mental tests.

9. *Deviation of Complement in Idiopathic Epilepsy.*—Using urine of epileptics as antigen, blood serum of epileptics as the source of the antibody, serum of normal rabbit as complement, hemolytic serum from a rabbit, suspension of human red blood corpuscles as indicator, Garnett came to the following conclusions: (1) Serum of epileptics contains some substance of the nature of a specific antibody. (2) Urine of epileptics contains very generally a toxin or substance specific to the antibody contained in the serum. (3) Serum of normal persons (not epileptic) does not contain this antibody. (4) The urine of some sane non-epileptic persons and some insane non-epileptics contains a substance which when mixed with serum of an epileptic, is capable of deviation of one and two

but rarely four minimal hemolytic doses—only 5 per cent. did so. (5) Urines of epileptic patients, when mixed in certain proportions with the serum of epileptics, are capable in a much larger proportion of deviating four minimal hemolytic doses of complement. In the second series of observations, 44 per cent. did so, in the third, 30 per cent. (6) The results are only striking when regarded comparatively, *i. e.*, when the positive results obtained with the epileptic patients are compared with the positive results obtained with non-epileptic persons. This method of observation, therefore, is of little value as a diagnostic of epilepsy. (7) The results of these observations are also of practical interest because if further study prove that some forms of epilepsy are of toxic origin, it should be possible to extract toxins from the urines of epileptic persons and to use these toxins as specific vaccine in the treatment of the disease.

(Vol. LVIII, No. 240)

1. Address in Neurology and Psychiatry, Australasian Medical Congress. W. BEATTIE SMITH.
2. The Classification of Insanity. JOHN TURNER.
3. The Development of Psychiatric Science as a Branch of Public Health. R. G. ROWS.
4. Some Points Complementary to the Institution of Post-Graduate Instruction in Psychiatry. DAVID ORR.
5. Sterilization from the Eugenic Standpoint, with Heredity Statistics. GEOFFREY CLARKE.
6. Some Statistics about Sterilization of the Insane. A. W. DANIEL.
7. Amenorrhœal Insanity. C. T. EWART.
8. Further Investigation on the Cerebro-Spinal Fluid in Insanities. G. SCOTT WILLIAMSON.

1. *Address in Neurology and Psychiatry.*—A discussion of some of the problems in psychiatry, especially the determination, if possible, of the causes of insanity, the importance of which is emphasized if success in the work is to be accomplished. The author calls attention to the probable overstatement of the alcoholic factor in view of the comparatively few pathological findings in asylums which may be traced to alcohol. An extensive statistical study of asylum pathological records is advocated to determine if possible the relation between the post mortem findings and the type of insanity. Such a study even if productive of negative results, would, in the writer's opinion, be of considerable value.

2. *Classification of Psychoses.*—Turner advocates essentially the present day classification, dividing insanity in two groups—1. idiopathic or hereditarily predisposed, embracing by far the larger number of individuals. 2. The traumatic or accidental.

The former he subdivides into three classes, (a) imbeciles, (b) dementia præcox, (c) acquired insanities, *i. e.*, epilepsy, delusional insanity, "lucid insanity" (meaning obsessions, psychasthenia, etc.), hysteria, affective insanity (manic-depressive), confusional insanity (exhaustion, alcohol, etc.), involutional insanity (mania, melancholia and dementia of this period).

In the second principal group are placed all cases of insanity arising from gross lesions of the brain, *e. g.*, infantile and senile cerebro-pathies, general paralysis, tumors and injuries.

3. *The Development of Psychiatric Science.*—Rows gives an account of the clinical and scientific departments of the psychiatric clinics in Ger-

many, calling attention to their importance and to the need of similar methods in England.

4. *Post-Graduate Instruction in Psychiatry.*—Orr writes on the evils of the asylum service in Great Britain from the assistant physician's standpoint and makes three recommendations. (1) Those who have decided to adopt the lunacy service as a specialty must be properly trained. Post-graduate courses have been considered and provided. (2) After a period of probation, say three years, asylum committees should consider an application to live outside, away from quarters, either in a house on the estate or in close proximity to the institution. (3) Every permanent assistant medical officer must have legal recognition.

5. *Sterilization from the Eugenic Standpoint.*—Clark has summarized his conclusions as follows:

1. Admitting inheritance to be the most important factor in mental constitution, it has yet to be shown that any practicable scheme of sterilization would materially diminish the normal increase of insanity.

2. We have no right to hold out a hope of material decrease from the statistics at present at our disposal.

3. There is urgent need of better record of family histories which should be kept separate from the present useless conglomeration which compose our statistics.

4. The suggestion is made that the chief danger from the eugenic standpoint is the large class of mental degenerates who are not insane.

5. The opinion is expressed that sterilization ought to be recommended in some cases of mental disease quite irrespective of the eugenic standpoint.

6. *Sterilization of the Psychotic.*—Daniel argues from the study of statistics of committed cases of insanity which after discharge had children, that only about 1.5 per cent. of total admissions to asylums would be prevented by sterilization of the insane before discharge. He is opposed, therefore, to sterilization as absolutely impracticable.

7. *Amenorrheal Psychoses.*—This is a small abstract of a paper which was published in the Transactions of the Royal Society of Medicine. The abstract is little more than a table of contents, and the original paper would evidently have to be read in order to ascertain the author's conclusions. In the extensive discussion which has been reported, it is clearly seen that many of those who heard the paper, were not in favor of the term "amenorrheal insanity."

8. *Cerebro-spinal Fluid in Psychoses.*—In a preliminary report of investigations undertaken to determine the reason for the discrepancy in the results when applying the Wassermann reaction to the cerebro-spinal fluid and blood serum of general paralysis the authors found and here record results significant in other directions. In these investigations it became necessary to detect the presence of blood-serum in the cerebro-spinal fluid and this was done by testing for a ferment normally present in blood-serum capable of splitting a synthetic di-peptide glycol-tryptophane into its two components. This ferment is not present in normal secretions such as cerebro-spinal fluid, synovial fluid, etc. A positive ferment test in cerebro-spinal fluid was obtained in cases of irritative conditions such as tubercular meningitis, tabes dorsalis, uremia, etc. Therefore, it seems probable that the presence of this ferment in cerebro-spinal fluid means an irritative transudation, though not necessarily inflammatory. In view of this, it is interesting to note that every cerebro-spinal fluid giving a positive Wassermann reaction contained the ferment. This

might indicate some underlying cause for irritation in such insanities which should be treated and not palliated. The authors also compared the Noguchi with the Wassermann test and found that they accord in 95 per cent. of cases. Attention is called in passing to severe after effects of lumbar puncture in certain young congenital imbeciles for which as explanation the theory is advanced of a possible patent ductus endolymphaticus.

W. C. SANDY (Kings Park, N. Y.).

Miscellany

REGENERATION OF MEDULLATED NERVES IN THE ABSENCE OF EMBRYONIC NERVE FIBERS, FOLLOWING EXPERIMENTAL NON-TRAUMATIC DEGENERATION. Elbert Clarke. (The Journal of Comparative Neurology, Vol. 24, No. 1.)

The present study is based upon experiments in which degeneration and regeneration of medullated nerve fibers were brought about under new experimental conditions. The results obtained relate, for the most part, to phases of the subject upon which the evidence has heretofore been incomplete. In this investigation, an experimental obstacle which has been responsible for the strikingly contrary observations between the supporters of auto-regeneration on the one hand and the advocates of an outgrowth of the axis cylinder on the other, has been entirely avoided. This refers to an ingrowth of foreign nerve fibers through the scar tissue into a regenerating medullated nerve. This obstacle was avoided by inducing degeneration in the peripheral medullated nerves of the domestic fowl by a prolonged exclusive feeding of polished rice, and subsequent regeneration by a return to an adequate nutritive diet.

The author shows that in the experiments described degeneration of medullated nerve fibers was brought about in fowls by a prolonged feeding of polished rice, and regeneration was accomplished by a return to an adequate nutritive diet. In such fowls the fibers are intact during degeneration and all traumatic and inflammatory effects produced by cutting the tissues and the nerve or of tying the latter are obviated; the process of degeneration can be stopped at almost any stage or greatly prolonged, and several stages of degeneration are to be observed in different fibers of the same nerve. In regeneration the possibility of an ingrowth of fibers from other nerves into the regenerating nerve under observation is eliminated and repair of the medullated nerves can be induced after any stage of regeneration. Ten to twenty per cent. of the medullated fibres of the nervus ischiadicus showed a complete fatty change of their medullary sheaths into globules of degenerated myelin and a segmentation or granulation of their axis cylinders. No multiplication of the nuclei of the neurilemma sheath could be observed and consequently no embryonic nerve fibers or Band-fasern. During recovery these degenerated nerve fibers attained new axis cylinders and the medullary sheaths returned to the normal. In other words, regeneration has been observed to follow degeneration in medullated nerve fibers without passing through the embryonic nerve fiber or Band-faser stage. By prolonging the degenerative process there resulted a multiplication of the nuclei of the neurilemma sheath. This and other experiments described tend to show that the embryonic nerve fiber may be coincident with a late stage of degeneration in medullated nerve fibers. It may not represent an early stage of regeneration and its presence does not

signify an attempt at regeneration on the part of the medullated nerve fiber.

In the absence of the embryonic nerve fiber, the degenerated myelin was absorbed with extreme slowness, persisting as droplets after 1 year and 14 days. On the other hand, where the embryonic nerve fiber was formed the degenerated myelin quickly disappeared from the fiber. The conclusion is reached that the proliferating nuclei of the neurilemma sheath participate in the resorption of the degenerated myelin. In regeneration a new axis cylinder was attained by outgrowth and in the absence of the embryonic nerve fiber. The new axis cylinder grew down the old medullary sheath which latter still contained large globules of degenerated myelin and fragments of the old axis cylinder. The outgrowing axis cylinder was seen to branch, and in cross-sections of the nerves two new axis cylinders were observed within the same old medullary sheath. The embryonic nerve fiber could, of course, play no part in the formation of the new axis cylinder either by auro-regeneration or by outgrowth.

No indications of regeneration were observed in the fibers of the spinal cord.

JELLIFFE.

THE STRUCTURE OF THE ROOTS, TRUNKS AND BRANCHES OF THE VAGUS NERVE. M. R. Chase and S. W. Ranson. (*The Journal of Comparative Neurology*, Vol. 24, No. 1.)

Most of this work was done on the dog, although the rabbit, cat, rat and man also were studied. The sharp contrast in structure between sympathetic and vagus offsets any close anatomical proximity. The serial section method was followed. The general results arrived at are summarized: (1) The various rootlets of the vagus and accessory nerves differ markedly in structure. The spinal root of the accessory is composed almost entirely of large medullated fibers with a very few small ones. The bulbar rootlets of the accessory are composed of large and small medullated fibers, with the small fibers predominating. These rootlets contain few, if any, non-medullated fibers. The rootlets of the vagus are of two kinds. Those of type I, probably efferent in function, are composed of many fine and fewer coarse medullated fibers. The medullated fibers are evenly distributed through these rootlets and there are few, if any, medullated fibers. The vagus rootlets of type II, probably efferent in function, contain large and medium-sized medullated fibers and fewer small ones. The medullated fibers are widely separated by enormous numbers of fine, non-medullated axons.

2. At the level of the upper part of the jugular ganglion the vagus and accessory nerves are fused into a common trunk in which it is possible to distinguish three areas derived respectively from the spinal root of the accessory, and the roots of the vagus. Each area presents the same histological characteristics as the corresponding roots, except that the fibers from the two types of vagus rootlets are now intimately mingled.

3. Below the level of the jugular ganglion the spinal part of the accessory which has maintained its independence throughout the common vagus-accessory trunk, now leaves it as the external branch of the accessory nerve.

4. The bulbar fibers of the accessory become intimately mingled with the vagus fibers at or above the level of the nodose ganglion. The so-

called internal branch of the accessory does not exist as a separate nerve, but is only a fascicle of the common vagus-accessory trunk.

5. While the vagus and sympathetic nerves are intimately associated in the neck, it is clear that no considerable part of the non-medullated fibers of the vagus are of sympathetic origin.

6. The pharyngeal branch is composed for the most part of large medullated fibers, but also contains a considerable number of medium and small-sized medullated fibers. It contains few, if any, non-medullated axons.

7. The superior laryngeal branch contains large, medium and small-sized medullated fibers, with the medium and small ones predominating. It contains non-medullated fibers in considerable numbers; but these are much less numerous than in the vagus trunk.

8. The recurrent nerve contains an area of large medullated fibers, destined for the larynx, and an area of medium and small-sized medullated fibers which are given off in its esophageal, tracheal and cardiac branches.

9. The pharyngeal, superior laryngeal and recurrent nerves take out from the vagus trunk almost all of its large medullated fibers, so that the vast majority of the medullated fibers in the thoracic vagus are either small or medium-sized. These are widely separated by non-medullated fibers.

10. The non-medullated fibers are present in much greater proportion in the thoracic portion of the vagus than in the upper part of the nerve. This is to be accounted for by the fact that a large number of medullated fibers have been taken away by the cervical branches, while practically all of the non-medullated fibers are carried down into the thoracic vagus. This increase in the proportion of non-medullated fibers in the lower part of the nerve is probably also in part due to pre-ganglionic fibers losing their myelin sheaths in their downward course.

11. Most of the medullated fibers in the thoracic vagus leave it through the bronchial and esophageal branches, so that the vagus as it passes through the diaphragm may properly be called a non-medullated nerve. It is composed almost entirely of non-medullated axons and contains only a few scattered medullated fibers.

JELLIFFE.

HYDROCEPHALUS, L. J. POLLOCK. (Journal A. M. A., Jan. 30, 1915.)

The author calls attention to the frequency of the association of hypopituitarism with chronic hydrocephalus. The causes of pituitary disturbances may be due, he says, to disorders of the gland itself, such as tumors, overgrowths or undergrowths, or they may be due to remote factors such as pressure caused by distant tumors and hydrocephalus. In severe hydrocephalus, the third ventricle, as well as the others, is distended with fluid and its cyst-like bulging presses on the underlying pituitary gland, the functions of which may be either depressed or exalted. In view of this fact, which is frequently shown in practice, its literature is scant. It has been noticed since by Marinesco and Goldstein in 1909, and Goldstein calls attention to the fact of its occurrence in an account of cases of hydrocephalus by other authors. Cushing also remarks on the scarcity of literature and other authors have given it more or less attention. Pollock refers to a previous article by himself, in which a case of dyspituitarism was reported by him, and the report of which is also given in this article. Since that publication in 1913, twelve additional cases have been observed,

ten in institutions and two in private practice. Three were described in addition to the one already mentioned, one of them rather fully, with an account of the postmortem observations. In all the cases, adiposity was the predominating feature, ranging from simple moderate overgrowth to actual monstrosity. Genital hypoplasia was observed but once, but all the patients showed a delicate pudgy tapering hand with a broad base. In six cases examined for increased carbohydrate tolerance, it was found, but not much importance is attached to this. Thermic reaction following injection of the extract of anterior lobe (Armour & Co.) was observed in several of the six cases described. The specificity of the adiposity described with pituitary diseases is not well established. Marburg first offered the theory that adiposity occurs not only in tumors of the pituitary but also in certain other cerebral growths of embryonic type, which cause it independently of their localization by virtue of a specific internal secretion. Pollock remarks that this is not altogether correct as is shown by the cases with pressure on the pituitary from hydrocephalus without tumor and from tumors of the pineal gland, not in the nature of a teratoma. Marburg subsequently recognized a tumor of the pineal gland with hydrocephalus, but in which the pituitary was not distended, though the infundibulum was involved. He formulates three groups of pineal gland disturbances: hyperpinealism, with obesity; hypopinealism, with genital atrophy; and apinealism with cachexia. Pearce Bailey and Jelliffe have summed up the literature and conclude that the whole situation needs more extensive study to determine whether pineal tumors alone can produce adiposity, apart from the third ventricle hydrocephalus. In the fully recorded case here noticed no conclusions as to the cause of the adiposity could be deduced, since both the hypophysis and the pineal gland showed disturbances from the dilatation of the third ventricle. In conclusion, Pollock calls attention to the similarity of serous meningitis to brain tumor and the possibility of pituitary symptoms leading to a wrong diagnosis of tumor of the hypophysis. The effect of a cystic third ventricle on the pineal gland deserves further study.

Book Reviews

STUDIES IN THE PSYCHOLOGY OF INTEMPERANCE. By G. E. Partridge, Ph.D.
Sturgis and Walton Company, New York.

This volume offers a new presentation of the problems of alcoholism and intemperance in a study brief and untechnical but based on wide knowledge and a fundamental insight into the nature of mankind. It departs from the ordinary ineffectual treatment of the outward symptoms of intemperance to deal with the real meaning and power of intoxication in human development, affording a true psychological basis for the control of this impulse and the direction of the force of which it is one expression.

The author's investigation begins even in the animal world. What is discovered there is more fully confirmed in an extensive study of primitive man and of early civilization, namely the relationship existing between intoxication and those states of excitement and exaltation that accompany the great racial functions, particularly reproduction. The intoxication impulse is at work as one important manifestation of instinct force proving its usefulness and necessity in the resulting increase of capacity for sustained excitement and a tendency toward that, which are essential to the creative activity upon which racial evolution depends. The impulse has manifested itself chiefly in religious activity and development. The excitement always demanded in this sphere has made intoxication appear pleasing to the gods or even divinely ordained, while many are the cults found throughout history dependent upon intoxication. Whether in the customs of widely scattered savage tribes or in the ancient Hindu soma worship; whether through stimulants the priest puts himself into the frenzied state necessary for communion with gods and spirits or the god is actually present in the wine, it is intoxication that produces the desired and effective religious condition. Out of these induced states on the other hand have arisen enlarged religious conception and quickening of faith in the supernatural. In its earliest uses moreover it was the accompaniment and source of a developing social consciousness, leveling natural barriers, producing good fellowship instead of original hostile relations and conducing to the establishment of guiding and restraining laws. For individual indulgence was not common among primitive peoples, rather intoxication was entered into by certain groups for special seasons and with definite purpose, and though great excess was the rule there followed regulated intervals of total abstinence. The very excess is a part of that deep-seated instinct to carry excitement to the point of abandonment to its highest culmination, which points to the fact that it is the state of intoxication that is sought rather than pleasure in the intoxicant.

In civilized nations again history testifies that a period of highest culture is preceded by intoxication expressing the intensity of the impulse for that excitement which, controlled and regulated, leads to the creative activity that marks the cultural period. This nature of the impulse makes intoxication likewise a characteristic of strong and dominant races. The narcotic side, on the other hand follows in a period of decadence and stagnation, when nations or individuals or certain groups of society can no longer turn the impulse to action but seek to escape the suffering inci-

dent upon inability to cope with the demands of existence and progress.

Literature and language testify to the important place of this impulse among all peoples. Medicine has witnessed through an exaggerated use of alcohol to the survival of the primitive conception of a life principle to be stimulated, an idea now modified and transformed and useful in psychotherapy in the principle of *aroused consciousness*.

Upon such well-laid foundation Dr. Partridge develops the practical side of his study. The individual manifests this impulse also as a part of the spirit of growth, that exaltation which must precede and accompany creative effort, and thus we find desire for excessive stimulation and the narcotic impulse following the natural periods of adolescent growth and maturer activity or of diminution of energy and interest. The excessive use of alcohol, now that its period of racial service is past, is abnormal and is found among two classes, those unable to rise above the level, where excitement fails to find its higher expression, and those so ill-balanced that they must seek narcotic effects.

The effect of alcohol on the individual can be studied by precise measurements and also by the examination of the feelings of the individual. In regard to the latter a large number of abnormal cases were examined by Dr. Partridge more or less fully according to opportunity and accurate laboratory tests were made upon normal individuals, ordinarily abstainers. The general conclusions agree. Physiologically alcohol is an injurious substance. It causes at first a temporary increase in physical and mental power but depression follows. There is however at one point a "feeling of power" in "intensity of consciousness" in which the individual feels himself freed from ordinary restraints and superior to ordinary limitations. This is allied to the social spirit which is so prominent in individual as in group intoxication and which is a predominant factor in the yielding to intoxicants.

It is upon an emphasis of its intrinsically social nature that the author would seek a cure for the prevailing evils of intemperance. So fundamental a craving, one which has served such broad racial purposes cannot be defined as sinful or immoral, nor can it be defined and eradicated as a physiological condition. It must be dealt with on broader terms. Education of the child and the youth for the guidance of his natural excitements into sustained and intensified activity through effective outlets, is the prophylactic need. The reeducation of the drunkard and readjustment of his surroundings so that new interests shall stimulate him and take up his energies, these will bring their own cure. Those opportunities for social contacts which the saloon has provided must not be neglected but there must be added what the saloon has failed to give, a "normal zest for life" and opportunities for real, effective activity.

This book is suggestive for such practical lines of endeavor based on a fundamental understanding of the problem with which it deals. It is stimulating too to further study and investigation into the deeper psychological nature of the social impulse bound with intoxication, the nature of the demand for the narcosis of alcohol, and further back the hidden relation between religious development and the intoxication impulse. Such are some of the problems to which this brief study might open the way.

JELLIFFE.

The Journal OF Nervous and Mental Disease

An American Monthly Journal of Neurology and Psychiatry, Founded in 1874

CELEBRATION OF THE THIRTIETH ANNIVERSARY OF THE PHILADELPHIA NEUROLOGICAL SOCIETY

FRIDAY EVENING, NOVEMBER 27, AND SATURDAY EVENING,
NOVEMBER 28, 1914

SCIENTIFIC MEETING

*Cadwalader Hall, College of Physicians, Friday Evening,
November 27, 1914, at 8:15 o'clock*

1. Charles K. Mills, M.D., Presidential Address: Concerning Cerebral Morphology in its Relation to Cerebral Localization.
2. Francis X. Dercum, M.D.: Nervous and Mental Diseases and the Newer Pathology.
3. James Hendrie Lloyd, M.D.: The Morphology and Functions of the Corpus Striatum.
4. Charles W. Burr, M.D.: The Psychology of Misers.
5. William G. Spiller, M.D.: Remarks on the Central Representation of Sensation.
6. Theodore H. Weisenburg, M.D.: The Founders and Work of the Society.

CONCERNING CEREBRAL MORPHOLOGY IN ITS RELATION TO CEREBRAL LOCALIZATION¹

BY CHARLES K. MILLS, M.D.

PROFESSOR OF NEUROLOGY IN THE UNIVERSITY OF PENNSYLVANIA; NEUROLOGIST
TO THE PHILADELPHIA GENERAL HOSPITAL; PRESIDENT
OF THE PHILADELPHIA NEUROLOGICAL SOCIETY

REMINISCENT—MORPHOLOGY IN THE EARLY HISTORY OF THE SOCIETY

This society organized in 1884 has made for itself a place in the history of American neurology. During thirty years it has never failed to meet at the appointed time. Its proceedings record much of the valuable neurological work, both actual and suggestive, of this period. As other decades roll on I have no doubt that it will continue to maintain its position. On behalf of the society I wish to extend our welcome and appreciation to those not of our membership who honor us with their presence this evening. This society has played a chief part in the building of what has been often spoken of in terms of compliment both here and abroad as "the Philadelphia school of neurology."

I have found the choice of a subject difficult and the necessary restriction as to time in presentation somewhat hampering. Two courses seem open, one, to give the results of a special line of neurological investigation, and the other, to present a subject of general interest, like cerebral morphology, referring among other things to the past work of the society in this field. I have deemed it best to choose the second of these courses and am more willing to do this knowing that several of my colleagues on the program this evening will give especial attention to the recording of the results of personal research. It is also somewhat the fashion for presidential addresses to deal with subjects of general interest.

On an anniversary occasion like this, one who has dwelt with

¹ Presidential address at the celebration of the thirtieth anniversary of the Philadelphia Neurological Society, delivered at the scientific meeting held Friday evening, November 27, 1914.

the society since its birth naturally turns back to the early period of its scientific history. Morphology was then one of the subjects especially interesting to some of our small band of neurological workers, among these being Dr. Andrew J. Parker who has passed from our midst and Dr. Francis X. Dercum who is fortunately still with us, fruitful in work.

My own attention and interest were fixed by the questions of cerebral surface topography, especially as illustrated in arrested, or aberrant fissures and gyres in the Caucasian brain and in the brains of other races as in the Negro and the Chinese. I have thought that lured by old memories and stimulated by recent tendencies in neurological research abroad, the subject of cerebral morphology in its relations to cerebral localization might prove of interest.

In our society and in the country at large, cerebral localization has been chiefly advanced by experimental physiology and clinico-pathology. A multitude of experimentalists have sought to locate functional centers and areas by focal extirpations, ablations, local freezings, and electrical excitation. Cerebrum and cerebellum, cortex and ganglia, grey matter and white, have been questioned with more or less success by these methods. Clinical medicine and neuro-pathology have had the most contributors. The symptomatology of circumscribed lesions, the projection, commissural, and association systems of the cerebrum, the cerebellum, and the basal ganglia, cerebral and cerebrospinal mechanisms,—these and other localization subjects have received the attention of our society. A critical examination, however, of the work done shows that omitting the first lustrum of our history to which I shall presently refer a little more at length, morphology, both macroscopic and microscopic, has been largely neglected.

As a society we would be furthering the best interests of neurology if our work were turned in larger degree to those morphological fields which are being tilled with such fruitful results by some of our confrères abroad.

Morphology is the science of organic form. In a broad sense it treats both of outer form and internal structural arrangement. While morphology in this fundamental meaning does not deal with function, the development of function in the organism receives its first and best elucidation in the study of anatomy and morphology. It is not so much that the form determines the function as that

the form is largely determined in the process of development by the function or intrinsic characteristics of the parts.

In strictness some of the data and inferences of this address may have reference as much to anatomy as to morphology, as it is not always easy in exposition to separate these two branches of biology.

Van Valkenburg (26) has well presented some of the most important principles which should guide us in the use of morphology as a groundwork for a doctrine of cerebral localization. As he indicates, the dividing lines which separate cortical localizations can be regarded in the first place as vertical to the cortex and in the second place as running parallel with it. The vertical lines of demarcation are represented by the fissures, and the parallel subdivisions by the cortical layers and strata. The two leading features in any fundamental scheme, on the one hand, are to be found in a discussion of the cortical areas bounded by the fissures demonstrably of functional importance, and on the other by a consideration of what is separated into circumscript regions by the cellular groupings in the strata or layers. Investigation of the subject from the morphological point of view therefore becomes a study of the place and plan of development of the fissures and their environing cerebral substance, and of the cyto-architecture in different locations of the parallel strata or layers of the cortex.

The first paper presented to this society at its first scientific meeting held in 1884 was one by me recording the history of the case of a murderer who was executed in this city, the contribution including an account of the fissural and gyral aberrations presented by the brain of this criminal. At the second meeting held on March 24, 1884, Dr. Andrew J. Parker made a verbal contribution on "Certain Primary Fissures of Fetal Brains," and at a later meeting after having studied with me the brain of another criminal, he described it before the society with some discussion by myself and others of its atavistic peculiarities, the brain being more remarkable in some of its reversions than any that have been published before or since.²

² During the delivery of the reminiscent and historical portion of the address charts were exhibited showing reproductions of photographs of a Chinese brain and the brains of several criminals and for comparison those of several eminent biologists described by E. A. Spitzka (25). Illustrations of the surface topography of the brains of lower and higher primates from drawings and photographs taken from the monograph of Parker were also exhibited.

DR. ANDREW J. PARKER, A PHILADELPHIA MORPHOLOGIST

Let me now call attention at some length to the personal history and to the main work of Dr. Andrew J. Parker (22), the distinguished cerebral morphologist who contributed to our early proceedings, feeling that to do this may not be without interest and value as it has always appeared to me that his work has not received the recognition which its merit deserves, that it has in fact to some extent been treated with unwarranted neglect.

Parker, who was born in Philadelphia and who was a graduate of the medical school of the University of Pennsylvania, early exhibited unusual interest in zoology and comparative anatomy and showed especial ability in the study of cerebral morphology. He was a favorite student of our greatest anatomist and biologist, Dr. Joseph Leidy. His one enduring contribution is that on "The Morphology of the Cerebral Convolutions with Special Reference to the Order of Primates." This was published by the Academy of Natural Sciences of Philadelphia in 1896, after his death which occurred in 1892. The fund required for this publication was contributed by his friends, the movement to bring this about having been initiated and carried forward by Dr. Dercum. The studies on which the paper was based were begun as early as 1877. Additions, extensions, and elaborations of his observations and theories were made until they assumed the form of the monograph as it was published by the Academy. The paper was awarded the Boylston prize by Harvard University in 1890. It is beautifully illustrated from original photographs and drawings by the author.

Let me here recall in summary a few of the main features of Dr. Parker's morphological monograph. He shows that only three conceivable theories can be advanced to account for the cerebral elevations and depressions. The first of these is the mechanical theory. According to this, the brain growing and developing with more rapidity than its bony environment, is necessarily thrown into folds and furrows in order to accommodate itself to this environment. This mechanical theory accounts for some of the fundamental primary fissures, as for instance, the fissure of Sylvius. According to the second view, the fissural and gyral development and arrangement depend upon a genetic principle. The third view, which is adopted by Parker, is a combination of both. According to this, certain of the fissures and

convolutions including the most important, are produced by merely mechanical causes, whilst others owe their origin to morphological processes of growth in the brain substance itself, the fissures representing lines of retarded and the convolutions of increased growth.

Starting with the idea of the development of the brain from the usually accepted cerebral vesicles, Parker shows that the cerebral hemispheres develop a hollow ovoidal body connected with the thalamencephalon, which body is arranged after a triradiate plan. This foreshadows the future lobes of the hemisphere. He distinguishes only three lobes, an anterior, an inferior, and a posterior. The anterior he terms the occipitofrontal lobe, which he describes as arching forward and descending to the position of the cerebral crus above. The second lobe, which he calls the occipitotemporal, arches downward and forward beneath the crus. The third or occipital lobe, passes directly backward. In other words, these three lobes are related respectively, one to the lateral ventricle and anterior horn, the second to the middle horn, and the third to the posterior horn. The arrangement is on a symmetrical triradiate plan, the fissure of Sylvius separating the parts anterior. He does not regard the insula as morphologically related to the other lobes of the hemisphere. Carrying out his hypothesis and in explanation of the entire morphology of the cerebral hemisphere, he believes that "the fissures and convolutions of the cerebral cortex are related in a regular and symmetrical manner to these hollow triradiate hemispherical bodies."

The first great triradiate fissuration is produced by a mesal arched fissure which divides into a superior or callosal portion enveloping the anterior horn and the lateral ventricle, and an inferior or hippocampal portion which corresponds to the middle horn, these being connected with the calcarine fissure which is the ectal correlative of the posterior horn. This triradiate fissuration occurs early in fetal life.

According to Parker the primary or typical fissures of the primate brain are the Sylvian fissure or fossa and the above described mesal arched fissure which consists of the callosal hippocampal and calcarine fissures. These fissures form the fundamental or plan fissures upon which the convolutions of all primate brains are based. They are not only the first to appear in the

development of the human brain and in all other primates, but they have also definite relations to the hemisphere itself. All of the other fissures and convolutions, according to Parker, are related in a definite manner to these primary furrows.

He holds that toward the end of the fifth month with the exception of the Sylvian fissure, the lateral aspect of the fetal brain is smooth, but on the mesal aspect the great fundamental triradiate fissure which is to dominate the future morphology of the entire cerebral surface is well developed. He differs from those who precede him and from his contemporaries in not according to other great fissures a similar importance. From his point of view the central, the intraparietal, the parallel, the first frontal, callosomarginal, and even the parieto-occipital fissure may be regarded as secondary, although the last has a special significance.

At an early date, about the third month of fetal life, the fissure of Sylvius divides the cerebral hemisphere into two lobes. In this fossa, broad at first, appears the insula. By the development of the temporal and especially of the frontal operculum the fossa is gradually closed in so that about the time of birth it is only a fissure, although even then the island of Reil is slightly exposed.

Parker's studies covered particularly the fetal brain, the brains of primates, and the negro brain. In the negro brain he found the insula usually more or less uncovered. In the brain of the white human being it is usually covered. The uncovering of the insula is not a characteristic of the brains of primates below man, or of man himself except in the case of the negro.

Shortly after the appearance of the mesal arched fissure, and in connection with the formation of the occipital lobe, the calcarine fissure appears as a nearly horizontal branch of the mesal arched fissure.

Parker divides the occipital from the parietal lobes by the internal and external prepeduncular fissures of the ape, or what correspond to them. He shows how, on the external and on the mesolateral surface of the hemisphere fissures more or less well and ill defined, demarcate this lobe. One part of this fissuration corresponds to the occipitoparietal fissure, the other to what is known as Wernicke's fissure which is often seen well outlined in low type human brains some of which have been described by me.

The parieto-occipital and Wernicke's fissure constitute for Parker the "occipital arch."

Parker divides the occipitotemporal lobe into four convolutions by means of three fissures, the first arching around the posterior extremity of the Sylvian fissure and the second around the same extremity of the parallel fissure, these being the well known first and second temporal convolutions. The third and fourth convolutions as regarded by Ecker, he seems to consider as one. He shows what is well known, that the longitudinal fissural differentiation is not clear between Ecker's third and fourth temporal convolutions. Parker's fourth occipitotemporal convolution consists of the union of the lingual and hippocampal convolutions. These fissures and convolutions bear certain distinct relations to the occipital lobe.

Describing the mesal surface of the hemisphere, Parker speaks of what he calls the midoccipitofrontal fissure which corresponds in large part to the callosomarginal fissure. He does not however regard it as stopping at the usual position according to the descriptions of Ecker and others, that is, at the posterior boundary of the paracentral lobule, but believes that it should be considered as extending backward until it meets the primary occipital arch on the mesal surface. It is true, as he says, that in many human brains an H-shaped triradiate fissure is to be found in the precuneus, and this or a portion of it he regards as a continuation of the usually-accepted callosomarginal fissure. He also points out, what I have observed, that the callosomarginal as usually accepted sometimes breaks up into two or more parts. This middle occipitofrontal fissure therefore follows in an archlike manner closely the course of the medial occipitotemporal fissure, and it and the convolutions bounding it develop according to Parker, on the same morphological principle as the latter fissure. The gyrus fornicatus, convolution of the cuneus, and hippocampal convolutions which together constitute the limbic lobe, form an arched convolution or series of convolutions following largely the course of the mesal occipitofrontal and mesal occipitotemporal fissures.

Parker does not regard the central or Rolandic fissure as a primary or fundamental fissure, like his triradiate fissure or the Sylvian fissure. It is, if all the primates be considered according to him, not as constant as the primary occipitotemporal fissure (first temporal or parallel fissure). It is absent or only slightly

represented in some of the lower forms of primates, but becomes more and more important as the scale is ascended and is usually defined and constant in the anthropoids and in man. In the latter it affords one of the best landmarks.

CRITIQUE OF PARKER BY DR. KAPPERS

I sent a copy of Dr. Parker's monograph to the eminent Dutch anatomist and morphologist, Dr. C. U. Ariens Kappers, director of the Central Institute for Brain Research, Amsterdam. Dr. Kappers made a careful study of the volume and with much courtesy wrote me two letters giving in condensed form the results of his study. I append one of these with a few unessential omissions in which Dr. Kappers, while criticizing some of the positions taken by Parker, shows his strong appreciation of our former colleague.

AMSTERDAM, September the 25th, 1914.

Dear Prof. Mills,—

Since my last letter to you I have continued to read Parker's paper and have now finished this lecture.

I am glad to have studied it again and more carefully than I had done, when preparing my report. I am much indebted to you that you have again called my attention to it. It is worth studying carefully.

Allow me to give you my impression of it and my criticism in the following lines:—

1. Concerning the transitory furrows as depicted in Parker's Fig. 2, p. 281, I believe with Hochstetter, Mall, Elliot Smith and others that they are artefacts. I have never seen them in my large collection of fetal brains, nor in fresh condition, nor after good fixation. It is not strange that especially these furrows—when they are formed, after a diminution of tension or putrefaction of the brain, should exhibit a mathematical arrangement, since they are dead lines not living lines. That in less good material a form as Fig. 2 can occur, is a fact which I could see in old preparations of the anatomical museum of our university. We may not however ascribe to them a fundamental character.

2. That fissures can be formed in regions where the presence of ventricles directly underneath the pallium diminishes the resistance is also my opinion and I would quote the same examples which Parker quotes, p. 288.

3. Parker's opinion about the cause of formation of the Sylvian fissure is also my opinion, especially *Manatus Americanus* is a striking proof for the fact that the brain-wall of the insula is fixed on the striatum by the number of fibers that pass through

the capsula interna and this fixed region is then vaulted over by the protruded frontal, parietal and temporal brain-walls.

4. Concerning the primary occipital arch (see Parker, p. 293 and further) I may remark that this conception holds good for the Simiæ.

Whether the simian fissure or perpendicularis externa of the simian brain may however be homologized with the perpendicularis externa sometimes occurring in human fetuses of the 6th and 7th month is doubtful.

There seems to be a good chance that the perpendicularis externa of such human fetuses is a product of impression of the sutura lambdoidea (hence Wilder's name "fissura lambdoida").

The simian sulcus or perpendicularis externa of apes may however occur also in adult man, as has been decisively proved by Elliot Smith. Prof. Davidson Black from Cleveland, who worked in my laboratory last summer, will publish soon a striking example of it.

5. I do not believe that Parker's primary occipital arch has anything to do with the transitory furrows of Parker's Fig. 2.

6. It may be true that in those animals, where Parker's secondary occipital arch (the Y-shaped fissure of Elliot Smith) occurs, this is a "vegetative reproduction," as he calls it, of the primary occipital arch; a repeated result of the same pressure-forces which caused the primary occipital arch. This would also explain why it occurs very rarely in man in a typical form, since also the primary occipital arch is rarely complete in man.

7. It is very interesting that Parker, p. 306, is inclined to derive the fissural system of the lateral convexity from horse-shoe-shaped fissures round the fossa sylvii. This principle has been proved to be right and is also used by Holl, Elliot Smith and myself. The three last named authors came however to a deduction different from Parker's. Starting from Carnivora (special Felidæ or Canidæ) we see in Ursidæ that the first lateral arch, the ecto-Sylvian fissure, has entirely disappeared in the insula, the first arcuate convolution forming something like the gyri longi insulæ in human anatomy.

In another carnivorous animals, *Lutra vulgaris*, we find sometimes the next process; the concealment of the anterior limb of the suprasylvian fissure (second lateral arch) and the anterior limb of the second arcuate convolution in the insula so that only the posterior limb of the suprasylvian fissure and of the second arcuate convolution remain on the surface forming the postsylvian fissure or fissura temporalis superior delimiting the temporal convolution. You find this process illustrated after drawings from nature (no scheme) in Fig. 5 of my London report.

The statement made by Parker that the intraparietal and inferior postcentral are derived from this arch is wrong (see Parker, Figs. 11 and 12, p. 306). You know yourself that the

intraparietal and inferior postcentral are never or very rarely connected with the temporalis superior.

In Carnivora as the Felidæ and Canidæ the third arched fissure is formed by combination of the coronalis (with ansata) and lateralis. From the coronalis and the ansata the central sulcus is derived and from the lateralis and its additional sulcus the medior postlateral, the intraparietal (with inferior postcentral) and the lunate or simian sulcus are derived. Parker has committed a mistake, I believe, in deriving the frontalis inferior from the anterior limb of the suprasylvian fissure and the frontalis superior from the anterior limb of the coronal fissure, but he is right in deriving the central from the coronal (plus ansata). My deductions concerning this point are found in my English report (Fig. 6).³

The frontalis inferior with the precentralis inferior is probably a derivative from the presylvian fissure of Carnivora and the frontalis superior is a new thing in the primates.

As far as concerns his mathematical conclusions I agree with Parker in the importance of Plateau's laws for the constellation of sulci. I believe however that though mechanical conditions have a considerable part in the formation of several sulci (calcarina, fissura hippocampi), a marked tendency to adapt themselves to functional (intrinsic) qualities of the cortex can be found in most of them. . . . I remain

Most cordially yours,

C. U. ARIENS KAPPERS.

P.S.—I will not omit adding that the reading of Parker's paper is a pleasure. The great knowledge of literature exhibited therein, the "causal thinking" (das causale Denken) which is so obvious on all the pages, then the broad viewpoint from which he considers these formations make his book stand out like a fertile piece of land between the sandy and incoherent literature on fissuration, which forms the greater part of communications on this subject.

SOME AMERICAN MORPHOLOGICAL OBSERVATIONS

I may be permitted at this point to say a few words additional about work done in cerebral morphology by Americans since the organization of our society.

To Professor Burt G. Wilder (27, 28) of Cornell University, Ithaca, N. Y., and to Professor Edward Anthony Spitzka (25), a highly gifted member of our own society, probably more than to any others either here or abroad is due the credit of emphasizing

³ A little part of the most frontal division of the anterior limb is perhaps represented by the sulcus opercularis, fissure Sylvius in apes.

the importance of studies in cerebral surface morphology as represented in high type human brains.

In my presidential address (15) at the meeting of the American Neurological Association held in Long Branch, June 21 to 23, 1886, I directed attention, with photographic and other illustrations, to the subject of the arrested and aberrant development of fissures and gyres in the brains of paranoiacs, criminals, idiots, and negroes. In the same address was incorporated a preliminary study of a Chinese brain, this investigation having been made in collaboration with Dr. Parker. In a journal known as *The Philadelphia Polyclinic*—at that time (September, 1886) the bulletin and organ of the post-graduate institution of the same name—I published an abstract of this address together with additional photographic illustrations of the brain of a criminal. This brain was one of the most remarkable ever studied by me.

My own observations on high-type human brains have been very few, one of these being made from photographs of the brain of Professor Alexander Winchell (17), the distinguished geologist, educator, and scientific author. In the morphology of his cortex one would look for the representation of high intellectual powers. Both his posterior and anterior association areas, so-called, fully carried out this expectation.

In my paper on Cerebral Localization in its Practical Relations (16), presented to the first Congress of American Physicians and Surgeons held in Washington in 1888, I called attention to the fact that it was important to remember the morphological variations and peculiarities in the human brain, especially in the posterior association area of Flechsig, when fixing the site for operation on the basis of symptoms presumably referable to lesions in this region, giving a number of instances of striking deviations from the usually accepted fissural and gyral pattern in human brains which had fallen under my observation.

MORPHOLOGICAL TOPICS OF SPECIAL INTEREST

Turning now to a study of the recent literature of cerebral morphology, let me bring to your notice several topics of special interest, namely,—(1) the significance of the fissures of the cerebrum and their relation to certain intrinsic localizations; (2) the process of tropism designated by Kappers as neurobiotaxis; (3) the value of cyto-architectonics in the study of localization; (4)

the histological anatomy and morphology of the basal ganglia especially of the thalamus; (5) the fissural and gyral morphology of the cerebellum; (6) cerebral anatomy, morphology and localization in their relations to mental disease; (7) the question whether cerebral centers so-called do in reality fully represent bodily functions; and (8) the anatomical and morphological material at the command of the society.

THE PHYSIOLOGICAL SIGNIFICANCE OF CEREBRAL FISSURES

Early in my morphological and localization studies I was impressed with the idea that the cerebral fissures or at least some of them were probably the boundaries of functional areas, arriving at this conception among other ways from a consideration of the atavistic peculiarities of low-type human brains, and the great difference easily discernible between these and the few examples of brains of individuals of higher intellectual capacity which I had opportunity to study either directly or in the literature of the subject. These low-type brains showed among other things, ape-like fissural reversions and tendencies to fissural confluences and the submersion of annectant gyres. The region of junction of the occipital, parietal and temporal lobes, which forms in its enlargement the posterior association area of Flechsig, was as strikingly simple in its fissural and gyral pattern as the same region was found enlarged and elaborated in the brains of men distinguished in various walks of life. The same relative simplicity and atavistic tendencies were found more or less defined in the midfrontal and prefrontal lobes, including the region of Broca.

Such observations while not sufficient for accurate localization determinations, were steps in this direction. They were helps toward a confirmation of the conclusions reached by Spitzka (25) in his interesting and important investigations. Speaking of the brains of distinguished men, convolutions according to Spitzka were found to be sometimes more massive, sometimes much convoluted; the subfrontal (third frontal) gyre was of unusual size and development; or this unusual development was observable in the frontal and parietal lobes. It was noticeable however in Spitzka's investigations that in some instances the brains of these distinguished men, instead of being very complex, were of unusual or comparative simplicity. Observations such as these, Spitzka correctly holds, point to the fact that much more than the mere

surface morphology of the brain must be taken into consideration in arriving at conclusions as to the value of lobar, gyral, and fissural markings and development, and that the quality of the cortex as indicated by histological research, and the amount, number, fineness or coarseness of the white fibers of the brain, are not to be lost sight of in our inferences.

Many of my own clinico-pathological observations recording focal lesions have seemed to me to indicate the boundary values of fissures, especially those of large size and depth. More than a quarter of a century ago I became pretty well satisfied that the central fissure was the boundary between the area for common and muscular sensibility and the motor region, expressing this view as early as 1888.

It is now very generally recognized that the central fissure forms this boundary. Similarly studies of actual cases with careful necropsies have seemed to me to indicate the frontier values of such fissures as the parallel, the Sylvian, the ascending Sylvian, the intraparietal, the calcarine, and even others of less size and extension like the precentral, the callosomarginal, and the retro-central. But this is a subject which can be properly elucidated only by elaborate consideration with the supporting data.

SIGNIFICANCE OF CEREBRAL FISSURES AND THEIR RELATION TO INTRINSIC LOCALIZATION (KAPPERS)

In the remarks which immediately follow I wish to call the attention of members of the society to some phases of the work of C. U. Ariens Kappers, of Amsterdam, doing this because of the intrinsic interest and great value of the researches of this distinguished Dutch anatomist and morphologist. The two lines of investigation of Kappers to which I shall confine myself are those relating to the significance of fissures and the doctrine of neuro-biotaxis.

Unquestionably one of the most valuable recent contributions to fissural morphology is the paper contributed by Kappers (8) to the Jubilee volume of *Le Nécrare* (Vol. XIV, 1913), dedicated to M. A. van Gehuchten, professor at the University of Louvain, on the occasion of the twenty-fifth anniversary of his professorship, on "The Significance of the Fissures of the Cerebrum in General and their Relation to Intrinsic Localizations in the Insular Region and in the Frontal Lobe."

Kappers first refers to the fact that Owen, Baillarger, Gratiolet and others had previously pointed out the difference between the abundance or number of convolutions in animals with larger and smaller brains (cerebrums). He uses his illustrations⁴ to show especially the greater convolutional development of brains of larger size. Calling attention to the law of Baillarger, that volumes of similar bodies are to each other as the cubes of their diameters, while their surfaces are to each other as the squares of their diameters, he shows that this law is modified when cerebral surface and cerebral volume are in question by the fact that the brain surface is thrown into fissures and folds. As in the main the thickness of the gray matter and its relation to the volume of the brain remain without much change, it becomes necessary that the cortex should be increased by the infolding of the surface in order to obtain a larger cortical area in animals of increasing size and importance.

Kappers differs with Jelgersma's idea that there is another explanation to be given, namely, a diminution of the white matter, which does not suffice to fill the pallium. He notes that, on the contrary, Brandis and Ernst de Vries have proved that the white matter of the cerebrum increases more in proportion to volume in large brains than does the gray matter.

Kappers shows that the law of Baillarger is modified in the ascending evolutionary series by the changes, displacements, and elaborations of the fissures, with the concomitant necessary alterations in the gyral masses related to these fissures. A study of Kapper's skilfully prepared chart shows the manner in which with increase of size and evolution of the intrinsic characteristics, these brains exhibit more and more fissural changes and elaborations.

Turning to the insula and its environment Kappers shows with convincing data that "the impartial comparison of the fissural and intrinsic qualities of the insular cerebral system teach us that there exists between them an evident parallelism of great importance and that the displacements of the fissural fields are not merely superficial changes, but they furnish interesting indications regarding the displacements in the mass of the cortex where the cerebral functions reside."

⁴ In illustration of this address a chart was exhibited, reproduced by permission of Dr. Kappers, this showing the difference between the convolutional and fissural development of animals with brains greatly varying in size.

Taking up the study of the fissures dorsad to the Sylvian fossa, Kappers then discusses the coronal, the pre-Sylvian, the ansata, etc. He shows how these develop in the ascending scale, changing position, form, size and connections. The coronal and ansata united correspond to the central in the ape. The frontal lobe develops step by step with the development and displacements, severances and unions of these fissures. This growth and development are due especially to the pre-Sylvian and not to the precentral field.

"More than is the case in the lower apes," he says, "the frontal lobe of the anthropoids and man—especially the part situated in front of the pre-Sylvian or precentral fissure—is excessively developed in comparison with other parts of the brain, and it probably contains fissures whose analogue does not exist in the lower apes. This summary suffices to corroborate our opinion that a submersion of a large cortical part in the insula must be explained by the enormous development of the frontal brain, especially of the field situated in front of the sulcus arcuatus, or its descendant, the inferior precentral fissure, the pre-Sylvian field."

Limiting his studies to the ungulates, carnivora, prosimians and man, Kappers brings out two facts,—the involution of the insular region, and the increase of the frontal lobes. As regards the former he confines his studies to the brains of apes and man, because he believes that he can best determine facts regarding such involution in these mammals.

He finds that in the animals below the primates, the Sylvian fissure has such heterogeneous variations that as compared with the primates one may speak of it best as a pseudo-Sylvian fissure. Three fissures constitute an arch around the Sylvian—the supra-Sylvian, the ecto-Sylvian, and the corono-lateral system. As evolution goes on this Sylvian field soon disappears from the exterior surface of the brain.

In the carnivora the ecto-Sylvian fissure becomes submerged in an increasing pseudo-Sylvian fissure, the convolutions also undergoing a similar involution. The supra-Sylvian fissure also undergoes submersion and changes in fissuration. The posterior part of the supra-Sylvian fissure which escapes submersion becomes the superior temporal fissure. At the same time, as evolution progresses the fronto-parietal portion greatly increases along the Sylvian fissure, becoming more and more opercularized.

In apes an orbital fissure is seen passing into the opercular region, and the insula begins to appear, but unlike the condition in man it is not covered. In brief, the study of the fissures and of the morphological characteristics of the brain shows a successive submersion and displacement of the furrows in the lateral region.

The question next studied is whether the intrinsic qualities of the frontal lobe show a parallelism with its fissural and general evolution.

Kappers recalls that the studies of such observers as Hitzig, Ferrier, Vogt and others indicate that in the carnivora a special localization of the movements of mastication, of the face, the eyelids, the head, etc., bears certain relations to the coronal and ansatal fissures. As the series is ascended the localization is still present, but is more discoverable nearer to the central fissure. The central fissure in other words now forms more exactly the posterior boundary than the ansatal and coronal fissures in the lower orders.

Electrical experimentations, says Kappers, confirm the opinion that the pre-Sylvian fissure corresponds to the arcuate because these two fissures form the anterior wall of the motor field of the head. In the anthropoid a nonirritable section is found in the neighborhood of the inferior pre-central fissure between this fissure and the true motor area which is further back toward the pre-central fissure. The chief electrically irritable field is along the central fissure.

Kappers further cites the evidence of secondary degenerations in favor of the theory of the motor field being entirely cephalad of the central fissure, as against former views that it was partly in the post-central region.

Evidence from the cyto-architecture of the frontal lobe confirms the fissural homologies and maintains the position that the intrinsic characteristics of the parts are related to fissural changes and displacements. The cortical layer in the ungulates which is limited by the coronal and ansatal fissures is almost confined to the agranular type. The internal granular layer (IV) is no longer seen but there is an abundance of pyramidal cells. Brodmann calls this field the pre-central agranular area. The characteristic cells in this region are the giant cells of Betz from which originates the pyramidal system of fibers. In the sheep this re-

gion extends as far forward as the pre-Sylvian fissure or nearly to it. Laterally it extends a little beyond the coronal fissure.

The very small part of the frontal lobe situated between the pre-Sylvian fissure and the sulcus rhinalis anterior which has a granular structure, does not belong to the pre-central region, but forms part of the frontal granular region in the primordial state.

In the carnivora, ascending in the scale, this pre-central region differs from that of the ungulates in that it extends more laterally (dorsally) even crossing at times the ansata in some carnivora like the cat and the bear. The field now begins to take a position which is more dorso-lateral and more oblique.

As we pass up in the carnivora the coronal fissure ceases to be the exact wall of the agranular pyramidal region, the motor field developing dorso-laterally to the fissure, not following it closely as a boundary. The pyramidal bundle arising from this new field is now more fully developed in the carnivora than it was in the ungulates. One dominating fact remains that the field has changed its position in relation to the sulci.

Summarizing his morphological facts, *Kappers finds that the results correspond with the data and conclusions of physiology, hodology, and cyto-architectonics. In the first place is noted a considerable enlargement of the frontal brain in phylogenesis. This enlargement is not due merely to the increase of the pre-central region in general, but especially to the expansion of that area situated in front of the pre-Sylvian, that is of the pre-Sylvian field of the frontal lobe.*

The learned anatomist of Tübingen, Brodmann, therefore is right according to Kappers, in dividing the frontal lobe into two parts, a pre-central properly so-called, and cephalad of this the frontal region. The latter is in the main what is usually regarded as the mid-frontal and prefrontal regions of the frontal lobe.

Kappers concludes that if it is true that the identity is not complete, if fissures must not always be considered as the limits or axial lines of the characteristic fields, the studies on fissural homologies are valuable enough to cause us to hope that morphology will continue to enrich us.

A general conclusion to which Kappers directs attention is that the cortex instead of increasing in thickness rather extends itself as a surface and therefore in order to maintain the original proportion between cortex and volume, takes numerous folds.

This is true even when we consider the question of stratification in its relation to function. The layers or strata which constitute the cortex increase with equal steps with the outside layer or extreme surface. If we look upon the question of function in its cellular relations, the same law holds good. The increase of the strata of the cortex through the process of gyral and convolutional development gives more room for cells of the kind which are needed for the functions acquired.

Kappers combats the idea that vascular relations, as has been held by many, determine the position of the fissures. While large vessels may follow the lines of fissures of considerable extent, and vessels may have a general tendency to follow fissural arrangement, we do not find these vessels placed at the bottom of the fissures, as would be likely if vascularization determined the position of the gray matter within these fissures. In the case of the cerebellum numerous fissures run transversely, while the great vessels pass longitudinally.

Summarizing his contemporaries, Kappers shows that Bolk, Kohlbrugge, and Stieda consider the sulci as regions of feeble growth. "Broca, Cunningham, Hill and Bolk presume that the sulci have a veritable physiological significance in that they delimit regions endowed with different functions. Ziehen likewise considers the sulci as arising from an intrinsic function of the cerebrum, but adds that they do not always follow the localizations. Campbell (4) and Vogt admit the possibility of an intrinsic homology, which however is never complete. Elliot Smith believes that the sulci were originally the limits (rhinalis, pre-Sylvian, supra-Sylvian, calcarina, anterior), or axial lines (fissura hippocampi, retro-calcarine), or areas of different function, but that the primitive character is effaced in consequence of cortical complications by associative centers in the sense of Campbell."

In other words, this last view is that while primitively the sulci may delimit areas of special function, an entirely homologous sulcus in a different animal may, because of special development, not so limit the primitive functional area. Brodmann holds that the cytoarchitonic structure of the cortex indicates that the sulci do not give exact localizations, although he agrees in a general way with the view of the fissural separation of functions. He believes that the effort to force these homologies into the service of the theory of structural centers is fruitless.

NEUROBIOTAXIS (KAPPERS)

A clear understanding of what is meant by that process of tropism which Kappers (7) has designated as *neurobiotaxis* gives a clew which can be obtained in no other way, as to the localization of cellular groups endowed with special functions. The principle underlying the arrangement and distribution of nerve cells and their processes, axones and dendrites, is one that has fixed the attention and attracted the close consideration of some of the most distinguished histologists of recent times and especially of His, Sr., and His, Jr., of Held, Ramon y Cajal, and C. U. Ariens Kappers. Some of the questions arousing interest are the following: what determines the direction taken by axones and dendrites in the process of growth and development? In other words, in what manner are cellular combinations accomplished and what determines the direction of commissural association and projection tracts? What are the laws governing the developmental arrangements of the nervous system? What is the basis of the organic form of special parts?

"I have used the term *neurobiotaxis*," Kappers says, "on account of its being a process of taxis or tropism occurring under normal conditions of nervous action, that is under the influence of reception and propagation of stimuli."

So far as I have knowledge of the subject, until the publication of the work of Kappers most attention was given to the question of the direction taken by axones and dendrites, by the processes of the cell in other words. His, Sr., regarded axones as taking their course along lines of least resistance, but this has seemed to me, as to Kappers, an explanation which does not explain.

Ramon y Cajal on the basis of an extraordinary amount of personal investigation arrived at his doctrine of chemotaxis. According to Cajal axones, dendrites, and collaterals grow as the result of the action of some chemical substance in the nervous system, taking this or that direction in obedience to this influence or by virtue of this influence in accordance with the irritations or stimuli from parts outside.

Kappers directed his attention to the question of the displacement of cell bodies as exhibited especially in the cell groups which

constitute the nuclei of the motor cranial nerves.⁵ Referring to his series which includes reptiles, amphibia, birds, etc., to man, Kappers shows how the cranial motor nuclei undergo a series of displacements, brought about through differences in the stimuli exerted on these motor cell nests by the sensory ganglia or nuclei. The motor nuclei are displaced to positions more dorsal or ventral, more cephalic or caudal, or to positions which are combinations of these caudal, cephalic, dorsal and ventral directions, and all in obedience to the changing and augmenting stimuli which are essential to the physiological processes characteristic of the species of animal under consideration. The general conclusion is that shiftings and displacements of the cranial motor nuclei are caused by differences in stimulation. This law is probably equally applicable to the positions taken by axones and their collaterals, by dendrites, and by the cell bodies themselves. Kappers believes that this neurobiotactical factor is found in the arrangement of the sympathetic system, while Cajal and Van Valkenburg see its influence in the arrangement of the granular layer of the cerebellum, and according to Herrick it can be equally traced in the architecture of the forebrain.

It is probable that when the facts of neurobiotaxis are thoroughly worked out for all parts of the cerebrum, it will be found that the motor areas have taken their position and form in close correlation with the sensory centers from which they receive their most important stimuli. It will be recalled by some members of the society that in one of my papers describing a new scheme of the zones and centers of the cerebrum, I expressed the conviction that every cortical sensory area so-called had closely related to it in position a motor cortical area for that particular sense; in other words that there is a visual motor, an auditory motor, a gustatory motor, an olfactory motor, and preëminently a cutaneo-muscular motor area. The last is the main cerebral motor area, that to which we usually restrict the term motor region. In other papers also I have argued that the cutaneo-muscular sensory area encircles, so to speak, the main motor region, and that it has subdivisions each of which is correlated with the subdivisions of the motor region itself,—a sensory subarea for the face, the head, the

⁵ In illustration of the subject of neurobiotaxis a chart was exhibited through the courtesy of Dr. Kappers showing the migration of motor nuclei in vertebrate evolution.

upper extremity, the trunk, the lower extremity, and probably for oral and visceral parts.

CYTOLOGICAL ANATOMY AND MORPHOLOGY

An investigator who has made important contributions to cytological morphology is Mott (21). One point in his work is salient,—that as vision advances, increase in the development of the pyramidal cells takes place. In the race and in the individual, development of the pyramidal cells both in number and size, increases step by step with the evolution of power. Mott refers to the well-known myelinization views of Flechsig, especially with reference to the optic radiations and the development of different layers of the visual cortex. The intermediate zones of Campbell (4) are but other names for the association areas of Flechsig. In the visual cortex the intermediate visuo-psychic area is the optic memory field of Wilbrand and Saenger.

In one of the charts exhibited to illustrate this address I reproduced from the work of Brodmann (3) a schematic representation of what this investigator believes to be the approximate distribution of functional areas in accordance with the results of his extensive studies on the cytology of brains of different orders of mammals including man. These results as here schematically depicted are largely, but by no means entirely, in accordance with those obtained by other methods of research. In this connection will be remembered the work of Campbell regarding the location of motor, sensory, and other areas as determined by cyto-pathological investigations.

In a general way the histological investigations of Campbell show that the posterior association area of Flechsig, the concrete concept area of the writer, is subdivided into subareas for the representation of concrete memories, those memories which are derived primarily by way of common sensibility and special sensation; they are, in other words, higher areas of vision, audition, common sensibility, olfaction and gustation.

Brodmann's results were not in all instances approved and corroborated by Ariens Kappers (9) who has written an admirable critique of the work. On the whole however the results obtained are strikingly in consonance with those which have been determined by other observers and by various methods of research.

Brodmann's book is in effect a study of the comparative organology of the cortex of mammals. Cytoarchitectonics, with which he deals, subdivides itself into (1) a reclassification of histological elements; (2) a stratification localization, and (3) a field localization, or topographical localization, the last being the only one considered by Brodmann as reliable. His first important contention is that for all mammals there exists a six-layer cortex, as follows: I. *Lamina zonalis*, molecular layer; II. *Lamina granularis externa*, external granular layer; III. *Lamina pyramidalis*, pyramidal layer; IV. *Lamina granularis interna*, inner granular layer; V. *Lamina ganglionaris*, ganglionic layer; and VI. *Lamina multiformis*, spindle-cell layer.

Brodmann holds, and is confirmed in this by Kappers's observation, that the neocortex of the lower mammals is also laid down in six layers.

Kappers in reviewing Brodmann differs with him with regard to Brodmann's manner of speaking of higher and lower animals too generally. What is more significant is the development of single layers and fields in connection with the special evolution of those sense organs, which are of particular importance in the life of the animal (Mott and Kappers).

Brodmann shows that a clearer view is obtained by grouping the six layers into two principal zones, the outer consisting of the first to third, and the inner of the fourth to sixth inclusive. The inner principal zone in lower animals is wider than the outer, which subserves higher and later acquired functions. This view, as Kappers points out, is strengthened by fiber anatomy, of which Brodmann takes but little account.

The second section of Brodmann's book concerns itself with the comparative surface organization of the cerebral cortex. He concludes from his examinations that the principles of field organization are similar in all the orders of mammals investigated.

The only thing determined with regard to the frontal region from experiments at present available on motor functions is that a closely connected agranular pre-central main region in the higher primates, causes fields with other functions to recede as has already been indicated in discussing the morphograph of Kappers on the significance of fissures.

Great merging and overlapping are shown in the post-central and parietal regions. This is what would be expected when it is

remembered that here in large part is the posterior association region or concrete memory field which represents special endowments in man. The studies of the brains of distinguished men are corroborative of these cytological findings in lower mammals.

The characteristic of the occipital region is the great widening of the inner layer. In the hedgehog Mott found this layer to be very stunted.

Kappers makes a statement of much significance in his critique of Brodmann, namely, "In order to see certain brain structures clearly in the light of evolution and correctly interpret them, we must begin with the simplest materials without classifying them backwards as heterotypical or rudimentary." The forms which Brodmann calls heterotypical are the fundamental elementary forms.

In the third part of his book Brodmann gives the morphological, physiological, and pathological cortex organology.

Brodmann as a result of his researches believes that in the cerebral cortex "a distribution of specific function among cells of different morphological character, different localization, and different importance, occurs."

Great as is this work of Brodmann to which I am able to make scarcely more than passing allusion, this distinguished neuro-anatomist himself has his limitations which seem to have prevented him from properly interpreting the entire significance of his own work. Kappers and Bolton (1), both careful students of the work of Brodmann, had recognized his tendency not fully to appreciate the value of histological research regarded both from the anatomical and morphological points of view as having convincing value in deciding questions in cerebral localization. For Kappers's view I must refer to his admirable critique of Brodmann.

Bolton speaks as follows: "This author (Brodmann) not only refuses to deduce physiological conclusions from his own anatomical facts, but denies that any proof exists of a correlation between functional significance and histological structure. Such an attitude on the part of a pure anatomist is, in an anatomical sense, to be commended on the ground that simple anatomical data cannot alone afford reliable evidence with regard to functional significance. It is nevertheless to be deplored that such a careful and accurate investigator of histological structure should extend

his generalizations beyond his own anatomical province and ignore the combined anatomico-physiological investigations of other workers in the field, on the apparent ground that the wildest of hypotheses have been suggested. That even the simpler processes of cerebral association are of very great complexity cannot be regarded in any sense as proof that the well-defined and constant histological structure of the cerebral cortex is not the anatomical counterpart of such processes. A similar line of argument applied to the bulb and cord would lead to the inevitable conclusion that the neurone complexes in this region of the central nervous system are also without special functional significance."

The histological anatomy and morphology of the basal ganglia—striatum, thalamus, hypothalamus, etc.—are attracting renewed attention from those interested both in fundamental biological principles and in the practical physiological questions like the localization of special functions. I must however pass by most of this work, referring my readers to the monographic articles of Ariens Kappers (6), Kinnier Wilson (29, 30), Malone (10, 11), and others. With regard to the problem of the functions of the striatum to which I have given some attention in several publications (18, 19) I can say only that the final settlement of the moot questions regarding the physiology of this great ganglion will in large part find their solution through studies in comparative cytology. One of the papers on the program of the evening is by our colleague, Dr. Lloyd, on the morphology and functions of the striatum. I am not prepared to endorse the view that this ganglion is a vestigial organ. While it is true that in the course of evolution it has assumed new positions in the cerebral mass and has undergone remarkable changes, it still retains in the human brain large proportions and a rich cellular endowment.

CELL TYPE LOCALIZATION IN THE THALAMUS

Malone working in Jacobsohn's laboratory, by special methods of investigating nerve cells for the determination of probable functions has thrown considerable light upon thalamic localization. According to this investigator there is probably in the thalamus not a single cell of motor structure, whereas in the hypothalamus numerous motor cells are present. This antagonizes the views of those who give any strictly motor functions to the thalamus, including the hypothesis of von Bechterew that

there is in a certain portion of this ganglion a motor mimetic center.

Let me refer briefly to the investigation of the thalamus by Malone (10, 11) as a valuable contribution by one of our countrymen to the relations of histological anatomy and morphology to localization. Malone's monograph is a continuation of the work of L. Jacobsohn on the nuclei of the human spinal cord and brain-stem which was published in the transactions of the Berlin Academy of Sciences 1908-09. It is intended to describe the cells of the human diencephalon (thalami and third ventricle). The effort is to group the cells of the diencephalon as separate nuclei so far as can be determined from their histological character. This work is not an attempt to divide the thalamus into separate regions as the result of various methods of biological research, but rather to make an anatomical and functional subdivision on the basis of histological investigations. Malone deals for the most part only with the authorities who confine their researches to the diencephalon of man, although he makes brief reference to the work of others, Da Fano and von Monakow, Haller, Ganser, Nissl, Münzer and Wiener, Bianchi and Cajal.

The method of subdivision of the thalamus adopted by Malone is that into groups of cells possessing identical histological character, completely disregarding the splitting up of gray matter by fiber masses into conventional nuclei.

Malone remarks that so far as he knows there is no work in existence which attempts to subdivide the diencephalon on the basis of purely cellular preparations. Referring to the histological observations of men like von Monakow, vonGudden, Forel, Meynert and others he says these were chiefly on the basis of carmine preparations which do not give as clear results in the way of cellular pictures as the method of Nissl. Malone warns the investigator not to regard the cell groups as separate nests or nuclei simply because these have been segregated by mechanical processes. In this category belong the cell masses which are pressed together by thick fiber bundles in their passage through the thalamus. This compression often causes considerable change in the number, position, size and form of the cells.

Malone endeavors to define clearly what he regards as cells of primary importance, considering them to be those groups in which the cells can be clearly defined from other areas by

markedly common histological characteristics. In arranging the cells he has considered as decisive their form, size, spatial relations of the cells to one another, and above all their structure. Changes in the first three characteristics are often caused by merely mechanical influences, and in such cases have only a superficial significance; if, however, these changes have a value for the nucleus they are nearly always associated with a change of structure. As characteristics of the cells he has considered the coloring and the order of the chromatophilic substance, the general sharpness of contours and of the inner structure, and the pigment contents of the cells.

Malone bases his observation on six series of the human adult diencephalon. For an account of his method of dividing and subdividing the thalamus I must refer those interested to his monograph.

First, warning again that groupings caused by mechanical means must be disregarded Malone makes some general inferences. He holds that we are justified in assuming that a more or less segregated group of cells which possess an identical cell character (above all the same structure) forms a primary nucleus which has a specific primary function, and further that two nuclei showing distinct differences in cell character also have different primary functions. That there exist inside of the primary nucleus parts which have relations to different fiber systems is no proof against the unity of the nucleus, as without such anatomical connections a working together of different parts of the nervous system is impossible. The same answer may be made to the objection that after a lesion of various parts of the primary nucleus the fibers can be followed into different parts of the cortex, or that after lesion of different regions of the cortex, the fibers can be followed into different parts of the primary nucleus; although such experimental examinations possess practical value they can only determine topographic areas which are correlated with other topographic areas, and they cannot have weight either for or against the unity of the anatomically conceived primary nucleus.

In addition to his research on the diencephalon, to which this reference has been made, Malone has done other histological work in anatomy and morphology (12, 13, 14) which has not only much absolute intrinsic value, but also is full of suggestiveness as to

the potentiality of his methods in the determination of the localization of cerebral functions. In his Johns Hopkins Monograph (14), he justly criticizes the neglect of a study of cell structure by most histo-pathologists. Cell preparations, as he shows, are too often made subsidiary to the study of tract degenerations, and little or no attention is paid to bringing the cell character into relation with a definite function. I cannot better emphasize his position on this matter of the neglect of cell types than by giving the following citation from his monograph in which is contained a reference to another of his publications: "The following instance is a good illustration of the value of carefully noting the cell character of various cell groups. In his excellent study of the dorsal (sympathetic) nucleus of the vagus nerve Molhant had shown that all the cells of this cell column give origin to all the vagus fibers which supply smooth muscle and heart muscle, and that certain portions of this cell column supply smooth muscle while a definite portion supplies heart muscle, but he did not attempt to show that a definite type of cell was involved in the innervation of each of these two types of muscle; after studying the vagus sympathetic nucleus I was able to show that the portions of the nucleus which supply smooth muscle and heart muscle may be readily distinguished by the fact that they are composed of cells of different types, and that just as heart muscle is histologically intermediate between smooth muscle and striated muscle, just so the cells of the vagus sympathetic nucleus which supply heart muscle are of a histological character intermediate between that of the cells which supply smooth muscle and striated muscle. Such an observation of the relation of cell type to cell function enables us to locate accurately a functional center even though its cells be mixed with those having a different function, and homologous centers may be recognized in different animal forms; at the same time it emphasizes the necessity of carefully noting the cell character of each cell group, so that even if the function is unknown the presence of a definite cell type will offer a problem for future experimental work, and in the meanwhile preclude the possibility of confusing this cell group with surrounding cells. These differences in cell type are of course most definite in the higher animals and especially in man, where the various cell groups are highly specialized."

In making his criticism, Malone says that he does so "not to

displace other methods, but to complement them and add to their usefulness."

With an additional citation from Malone's conclusions I must content myself by referring to his bibliography appended to this paper.

"The histological character of a nerve cell is an indication of its function. Differences in connections with portions of the organism which differ merely in spatial relations do not involve a difference in the character of the nerve cells, but are associated merely with the *location* of the nerve cell; for instance, arm and leg muscles, flexors and extensors are all innervated by the same type of cell, although such differences in peripheral connections correspond to the differences in the position of the corresponding nerve cells. It is therefore evident that experimental work which determines the connections of various portions of a given region with different portions of the organism, without taking into consideration differences of cell character, fails to distinguish between differences of connection dependent merely upon *spatial* differences and those differences of connection which involve differences in *cell activity*, such cell activity being indicated, not only in the nervous system but in all portions of the entire organism, by a definite type of cell character."

CEREBELLAR FISSURES AND CEREBELLAR LOCALIZATION

In the last decade investigations in fissural and stratificational morphology have been applied to the study of cerebellar localization as illustrated by the mammalian schema of Bolk (2) and the physiological schema of von Bechterew, based in large part on Bolk's researches. Studies in comparative anatomy show that some of the fissures in the human cerebellum which seem on first glance because of their depth, width and length to be of the greatest physiological importance are found on closer investigation not to have this value. The great horizontal fissure (*fissura horizontalis magna*), for instance, is of less importance as a physiological boundary than the crescentic fissure of the human anatomists. This, usually called the primary fissure (*sulcus primarius*), is from the standpoint of comparative anatomy of fundamental morphological and functional importance.

In a recent paper by Dr. Weisenburg and the writer (20) attention has been directed to this matter of the significance of fis-

sural development in establishing fundamental facts regarding cerebellar localization, a subject to which Rothmann (23) in his great papers on the cerebellum had previously given much attention in connection with the description of his own physiological researches.⁶

The primitive morphological subdivision of the cerebellum is into an anterior lobe comparatively simple, and a posterior lobe of much complexity, these being separated by the so-called primary fissure. As the mammalian series ascends to man the most striking and readily demonstrated change which takes place is that represented by what Bolk has called the monstrous development of this posterior complex lobe. It is in this part of the cerebellum that the vermis first takes on a really separate form and here appear new fissures and convolutions of particular shape. The development of this region is the result of extraordinary synergic acquisitions in the higher primates and especially in man. It follows, therefore, as a fundamental fact in localization that in this part of the brain must be represented those functions of synergy which are the particular possession of man—his ability to stand erect, and his ability in this position to perform the innumerable skilful synergic movements which are his particular attributes. Through such elementary facts we arrive at points of departure for studies of cerebellar localization.

As has already been indicated, the influence of neurobiotaxis in the development of the cerebellum has been studied by Cajal and van Valkenburg, and investigation of the cytoarchitecture of the cerebellum will eventually play an important rôle in the accurate determination of its functions.

HISTOLOGICAL ANATOMY AND MORPHOLOGY AND MENTAL DISEASE

Clearer and more rational views regarding the classification, symptomatology and diagnosis of mental diseases can, I have long believed, be obtained only by our giving closer attention to questions of anatomy and morphology and especially of histological anatomy and morphology.

Bolton, in a recent book on "The Brain in Health and Dis-

⁶ Charts were shown illustrating fissural plans in different orders of the primates and also Bolk's scheme of the mammalian cerebellum and a tentative scheme of physiological areas and centers in the human cerebellum.

case" (1), shows a full appreciation of this fact. The mazes of doubt and uncertainty regarding the underlying pathology of dementia præcox, paranoia, mania, melancholia, paresis, epileptic insanities and other forms of mental disease and disturbance, are recognized by most workers in the field of psychiatric neurology.

Bolton holds that "there exists a definite structural basis for the special symptomatology presented by the subjects of mental disease, and perhaps also for the unequal mental endowments of sane or normal persons." His investigations in demonstration of this thesis are micrometric, that is he measures with microscopic exactness the depth or thickness of different cortical laminæ in chosen positions in the brain in different forms of mental deficiency and disease—idiocy, imbecility, paranoia, etc.

A study of the evolution of cortical lamination in the fetus and in the adult, Bolton remarks, is confirmatory of certain now generally accepted ideas as to differences between the projection cortex and that of so-called higher association areas or zones. This lamination has been traced by him step by step in the cases studied with microphotographs and micrometric measurements, from the state of undifferentiated neuroblasts in the early months of fetal life, to the laminated appearance at birth, in childhood, in youth, and in the adult. I cannot here make more than the briefest reference to such investigations and their results. Some points are of fundamental value, as for instance, the fact that the innermost laminæ assume their form and full development earliest, the outermost last. When such projection areas as the visuo-sensory are considered and these are compared with the visuo-psychic, and with the prefrontal or higher psychic region, it is evident that the first receives its most complete development earliest, others later, the prefrontal being the last of the neopallium⁷ to take on its full laminar appearance.

The psycho-motor area has its evolutionary periods as regards lamination, as have all the rest. The generalization which becomes evident in the application of cytological methods to the

⁷ The neopallium is a term applied by Elliot Smith to the cerebral cortex peculiar to the mammalia. It consists of three laminæ which undergo in different members of the scale and in different regions of the cerebrum, varying degrees of evolution or suppression. The neopallium, which roughly consists of the entire cerebral cortex with the exception of the pyriform lobe and the hippocampus, differs from the archipallium, or the remainder of the cortex, in the possession of an outer or pyramidal cell-lamina, the middle and inner cell-laminæ being common features of the whole of the cerebral cortex. (Bolton.)

explanation of psychic acquisitions, is that the lamina most essential to the existence of the individual is that which is first and most organized, while those which represent the higher and the highest intellectual acquisitions are the later and latest to be fully organized. Such facts as these, Bolton and others have shown, can be applied with scientific value to the explanation of mental disease, especially to those disorders of the mind which are of embryonal or teratological origin. Herein we see a fruitful field for the study of localization in connection with both physiological and clinico-pathological interpretation. One point of much importance, that in the prefrontal region laminar evolution not only occurs later than in other regions as already stated, but is individually variable might also here be emphasized that fissural evolution has close analogies as regards both time and individuality, with the evolution of lamination.

The main idea in this address has been to emphasize certain morphological and anatomical facts and principles, a knowledge of which is fundamental to a comprehension of the subject of cerebral localization. I might here with van Valkenburg ask the question—do the so-called centers, cortical and subcortical, in reality represent bodily functions? While it might be necessary to answer this question, baldly put, in the negative, it nevertheless remains true that cerebral localization is a reality. It is only necessary that one shall understand clearly what he means when he talks of centers and zones and their functions.

Experimental physiology and clinical medicine united with pathology would seem to place the existence of definite centers and zones beyond all doubt. It is indeed true that extirpation of a circumscribed portion of the cerebral cortex in this or that part of the brain often results in the production of certain well defined symptoms. It is equally true that irritation by an electrical current or neoplasm may bring about definite phenomena. The one point of interest in this connection is that while centers are sufficiently defined to enable the neurologist to help his friend the surgeon to the performance of operations, even the neurologist of largest experience and acquisitions does not know the function of any single part of the human brain in its entirety until he has a thorough knowledge of all the connections of that part. In other words, he must through studies in anatomy, morphology, clinical medicine and pathology be able distinctly to recognize that a

given so-called center—one for example in the motor zone—has a complex of functions which can only be determined absolutely by a knowledge of all its connections, projection, associative, commissural, etc., with other parts of the nervous system. Even in the case of a motor center the mere understanding of the phenomena of movement which it represents is not sufficient. The diaschisis phenomena of von Monakow can be clearly revealed only by an elaborate consideration of all the cellular connections of any given region.

It has always seemed to me that J. Hughlings Jackson (5) more clearly than anyone else had a true vision of the inner meaning of the idea of localization in the cerebrum and indeed in all parts of the nervous system. In the highest sense of the word he was a localizationist and believer in the doctrine of "centers," but for him centers had a significance not fully grasped by others. In his fundamental conception lay the germs of the hypothesis and theories which since under various names have been brought to the attention of neurologists, as for instance, the doctrine of neural mechanisms and of diaschisis of von Monakow. Jackson's lower, middle, and higher levels in the nervous system are levels of representation, re-representation, and re-re-representation. The idea is simply that of the most differentiated representation correlated with higher and highest representation of the same parts in more and more complex combinations or syntheses. "I have already said that units of the highest centers," says Jackson, "are at once universally (factor integration), and specially representing; that every unit represents all parts of the body, and yet that each represents some one part of it first and most. This is repeating that the peculiarity of every part of the body is respected, however high evolution is carried. The fact that a man can shrug his shoulder of necessity implies that the movements effecting this operation have, in some parts of the highest centers, a more special representation than all other movements have in that part. In so far as any part of the body (lowest level of evolution of the whole organism) has a degree of independence of the rest, in so far it has a corresponding degree of speciality of representation in the highest centers. I hope these remarks will be borne in mind by those who object to the use of the term 'centers'; they will see that I do not use it rigidly. Thus the 'arm center' in the Rolandic region (middle motor centers) is

for me a center representing movements of all parts of the body, but yet movements of the arm very much more than any other movements."

AVAILABLE ANATOMICAL AND MORPHOLOGICAL MATERIAL

In conclusion it may be asserted that the society's record of work has been one of which it may justly be proud. Nevertheless it has not done what it might have accomplished in the fields to which I have directed attention this evening. This is not because we are unprovided with the material for such work. It is at our hand.

In the first place, the Zoological Garden of Philadelphia affords constant opportunity for obtaining the brains of animals of different species. I have here tonight a series of brains obtained through the courtesy of Dr. Williams B. Cadwalader, one of our university neurological staff who is also prosector at the zoological garden. These specimens illustrate a series of mammalian brains from the lemur *negrofrons* to the chimpanzee. This series with other specimens from lower animals which could readily be added to it affords material for comprehensive and thorough morphological investigations, both macroscopic and microscopic, similar to the researches of Parker and of our confreres abroad. Human specimens, especially fetal brains and low type and average adult brains such as can be obtained from the Philadelphia General Hospital, are always at our disposal. We still have in the laboratory of neuropathology at the University of Pennsylvania some specimens of the brains of criminals and paranoiacs which were used many years ago in preparing one of my papers. If anyone should object to these specimens that they are antique for purposes of real research, I might refer him to the morphological work of Elliot Smith (24) on the brains of Egyptian mummies. The story of his work is by the way an interesting one and may serve to lighten the more serious strains of this paper.

Elliot Smith was for many years professor of anatomy at the Egyptian Government School of Medicine, at Cairo. While in Egypt he made a particular investigation of the brains of a number of mummies of early historic and pre-historic times. In one of his papers he has given photographic reproductions of the fissures and convolutions of some of these specimens. It was urged

against the reliability of his investigations that Virchow and others had indicated their belief that in the mummies of Egypt the brains were not preserved, but that the substance found in the skull was a resinous material used in the preservation of the mummies. In answer to this assault upon the accuracy and reliability of his observations, some archeological inquiries of much interest were made.

Elliot Smith estimated that in the forty-seven hundred years during which the Egyptians practised embalming, about four hundred millions of people were embalmed. He says also that almost every archeologist who has studied the bodies found in the innumerable cemeteries of Egypt knows that the brains are frequently present in the skulls of those bodies not mummified by artificial conservative processes. His investigation shows that the materials found in many of the crania submitted for inspection were undoubtedly brains. In addition this is proved by the fact that such masses have been obtained in crania which have not been severed from the body and which in fact have not been opened. These intracranial masses are brains which have been dried and preserved by natural processes. In one prehistoric cemetery containing nearly five hundred bodies the brain in every instance had been preserved. The same has been noted in other cemeteries of later periods, especially when the bodies have been preserved in dry soil and have been placed where the air had no direct access to them. He found that the brain masses varied much in size. They were usually about two thirds or less than half of the original dimensions. In the case of many of these masses it was possible accurately to determine the course of every sulcus on the lateral and mesal aspects of the cerebral hemisphere. He gives actual photographs and two diagrammatic schemes of the fissures and convolutions in a specimen from a cemetery of the XVII dynastic period (about 2000 B. C.). It would appear that the process of embalming with the removal of the brain was confined to those who had sufficient influence or left sufficient money to have this semi-religious rite performed. In these cases or some of them it was said that the ethmoid bone was fractured and the brain was sucked out by some method of aspiration. It would seem, therefore, that the kaisers and mikados of those periods, known as pharaohs, and the Rockefellers, Carnegies and Dukes of West-

minster of Egyptian times probably had their brains removed in the process of artificial conservative mummification; while the mass of the people, fortunately for the cerebral archeologist, were buried brains and all, and if these burials were in soil sufficiently dry and well drained the specimens remained fit material for study after thousands of years.

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NERVOUS AND MENTAL DISEASES AND THE NEWER PATHOLOGY

By F. X. DERCUM, M.D., PH.D.

PROFESSOR OF NERVOUS AND MENTAL DISEASES, JEFFERSON MEDICAL COLLEGE;
CONSULTING NEUROLOGIST TO THE PHILADELPHIA GENERAL HOSPITAL;
FOREIGN CORRESPONDING MEMBER OF THE NEUROLOGICAL SOCIETY
OF PARIS; FOREIGN CORRESPONDING MEMBER OF THE NEU-
ROLOGICAL AND PSYCHIATRIC SOCIETY OF VIENNA;
MEMBER OF THE ROYAL MEDICAL SOCIETY
OF BUDAPEST

The time, it seems to me, has arrived when it is possible to assemble and to weigh as to their significance numerous important facts bearing upon the pathology of the nervous system. In order that we may view these facts intelligently, it is necessary first to dispose briefly of the following preliminary considerations. To begin, nervous diseases are quite commonly divided into organic and functional. This distinction possesses a certain amount of practical value, but when analyzed it proves in its essence to be neither valid nor scientific. An affection which is attended in its early stages by no organic changes whatever may in a later stage be attended by changes gross in character. A notable instance of this is paresis, the symptoms of which may be pronounced in the early stage and yet this early stage reveal not the slightest structural change in the nervous system. Again, a broad distinction is habitually made between nervous diseases on the one hand and mental diseases on the other. While this distinction has in its turn an undisputed practical value, it likewise proves upon analysis to be invalid. This is shown by merely a cursory examination of the facts. Thus, it is well known that epilepsy may in addition to, or in place of, the gross nervous phenomena which it ordinarily presents, also present mental phenomena, and, indeed, often to such a degree as to necessitate its being classified among the insanities. A similar truth obtains in regard to hysteria, and when we turn our attention to its congener psychasthenia, we at once recognize that we are dealing with an affection which is still, in large part, in the domain of

nervous diseases and yet frankly occupies a place among mental diseases. Similar truths obtain in regard to melancholia and mania, and dementia præcox and paranoia, and, indeed, to mental diseases in general. The latter are, properly speaking, in this sense, merely functional nervous affections and must be embraced in any inquiry dealing with the pathology of the latter.

The facts which I am about to present in such an inquiry are facts which deal with material things. This truth cannot be sufficiently emphasized. The facts deal with changes of structure and function, with problems of metabolism, intoxication, infection, with questions of biochemistry and with other matters, all of which are identical in their nature with those which confront us in internal medicine. It is a remarkable fact that for years past many alienists have turned their attention to psychology rather than to internal medicine, the facts of which though of ever increasing importance have received but scant attention at their hands. The day however has gone by when we can be content with explanations of psychology and least of all with those of that now fast vanishing cult psychoanalysis.

All of the phenomena presented by the human organism, digestion, nutrition, the maintenance of the bodily temperature, the elimination of nervous and muscular energy, are convertible into terms of tissue chemistry. It is the summation of these manifold and complicated chemical processes that constitutes that moving aggregate which we recognize as the living individual. It is the derangement of these processes which constitutes disease,—a derangement which may have its origin spontaneously in some fault of the organism or which may be secondary to some cause introduced from without, such as a poison or infection. The metabolism of the body in health is as yet, it must be admitted, very imperfectly understood; notwithstanding there can be no doubt that its disturbances entail states of intoxication, and in these states of intoxication must be sought the pathology of mental and nervous disorders. Theories of autointoxication are as old as Hippocrates. In modern times they were advocated by Pinel, Esquirol, Morel, and others, though these older psychiatrists were inclined to regard states of intoxication as secondary to a primary affection of the nervous system. Numerous subsequent observers, among whom should especially be mentioned

Mendel, studied the urine, blood and other fluids, but while many of the facts ascertained were interesting, nothing of value was achieved; and, indeed, the subject did not assume even a scientific character until Bouchard introduced his theory and methods. Bouchard taught that the organism was a laboratory of poisons and this equally in the normal as in the pathological condition. In the normal condition these poisons are in one way or another made harmless, but in pathological states, when the protective measures of the organism are weakened, these poisons circulate in the blood and are excreted by the urine. Bouchard it will be remembered worked out a method of determining the degree of the toxicity of the urine, a method which found a later application also in the study of the toxicity of the serum. A definite amount of normal urine was found to be lethal to a certain weight of rabbit and thus was determined the coefficient of the toxicity of the urine. As the urine of the insane frequently showed an increase in this coefficient it was naturally ascribed to an increased output of poisons. However, the existing knowledge of physiological and chemical processes was not sufficiently advanced to render the results of value. No information was yielded as to the sources of the poisons and but little as to their character. Besides the method took no account of such an important factor as the difference between the osmotic pressure of the urine and that of the serum of the blood; and the findings were also confusing in that the coefficient sometimes rose with a maximal intensity of symptoms and sometimes rose with a minimal intensity, or instead of rising, falling under the same circumstances. These facts, while they admitted of explanation, did not do so in a satisfactory manner. Similar experiments were made and similar results achieved with blood serum, and in this connection Rebicci's observations that the blood of epilepsy, paresis, senile dementia and amentia is poisonous to the leech, are of historic interest, as are also other observations made as to the toxicity of the perspiration and of the cerebro-spinal fluid.

I well remember the profound interest which these theories of toxicity aroused, as likewise the disappointment which necessarily followed. We all of us remember, also, the extensive rôle that was at one time assigned to the uric acid group and its congeners, a rôle that to-day still demands consideration. The

leucomaines, as they were named by Gautier, are regarded, as is of course well known, as the products of the incomplete oxidation and hydration of proteid substances. In some respects they resemble the vegetable alkaloids, and it was at first believed that their presence in the urine was sufficient to account for the symptoms of a given case. All that it seemed necessary to do was to further the elimination of these poisons, or to prevent their formation, or in some way to render them harmless. These expectations were of course doomed to disappointment, but, though the doctrine of the leucomaines like the earlier views of toxicity failed of a definite result, the one great truth still remained, a truth which Bouchard and his followers had early perceived, namely the great fact of auto-intoxication.

The doctrine of auto-intoxication gained force from the subsequent and rapidly accumulated knowledge as to the rôle played by poisons of exogenous origin in the production of mental disease, notably alcohol and the infections. The disorders which these poisons cause,—delirium, confusion, stupor, dementia,—are now clearly recognized. Regis and later Kraepelin pointed out the rôle of the infections. Kraepelin was one of the first to insist that the mental symptoms are the result of the action of bacterial toxins. These toxins, it would appear, may act directly upon the cortex, or the infection may bring about disturbances in the functions of other organs, disturbances leading to changes in the general metabolism of the body, to the formation of endogenous toxins and to the consequent auto-intoxication of the entire organism. Infections variously involve the liver, the kidney, the thyroid, the adrenals and other glands and tissues. The fact of such involvement is based upon indisputable clinical and pathological evidence. That under such circumstances an intoxication may be very complicated and very persistent goes without saying. A proper appreciation of this fact lends renewed interest to the mental phenomena of infection. The prolonged confusions that now and then occur during the post-febrile periods of typhoid fever and other acute infections, the prolonged confusional insanity every now and then met with in alcoholic subjects, are clearly due not to the original infection, not to the poison originally ingested, but to the secondary toxemia of a deranged metabolism. Again, the part played by the in-

fections enables us to understand more clearly the mental phenomena every now and then observed in the early stages of the subacute infections, syphilis and tuberculosis. Here mental symptoms are often observed long before the physical signs peculiar to the infection become manifest, *i. e.*, long before organic changes occur. Such symptoms must necessarily be toxic in origin. That they occur early in paresis and early in tuberculosis every alienist knows.

With these considerations before us, let us turn our attention to auto-intoxication proper, that is, to primary or endogenous poisoning. A moment's reflection reveals that primary auto-intoxication is of two kinds; first, one that is gastro-intestinal in origin and another that arises in the tissues of the body itself. This distinction, however, may in practice be a very difficult one. An intoxication by way of the intestinal tract may give rise to secondary disturbances of metabolism within the body, *e. g.*, derangements of the liver or of the thyroid gland, or, intoxications having their origin within the body proper may be followed by a secondary intestinal intoxication; of this melancholia and other affections in which the innervation of the intestinal tract is diminished, afford abundant examples. In considering gastro-intestinal intoxication as a possible primary factor, we must bear in mind that in the normal or healthy individual, intestinal poisons are destroyed by the liver, the thyroid and by the other glands and tissues generally, or, eliminated by the various excretory organs. Poisoning of the nervous system is thus effectively guarded against. However, in diseased and defective states, it is possible that invasion occurs and that it may also produce, as above pointed out, secondary disturbances of metabolism. The presence of phenol, indol and skatol in large amount bespeak, of course, intestinal intoxication, but, on the whole, it may be safely said that when there are present marked or persistent nervous symptoms, we have to deal with a coëxistent and probably primary, interstitial or endogenous poisoning.

The detailed chemistry of the urine, a chemistry which deals largely with the end results of the disorders of metabolism, cannot long detain us here. The amount of nitrogen, sulphur and phosphorus excreted, the mineral waste and the output of water are all facts of great interest but are obviously secondary in im-

portance to other and more recondite changes. Suffice it to say that some of the facts suggest disturbances of the glands of internal secretion; others indicate relationships between different disorders, e. g. between migraine and epilepsy, between epilepsy and diabetes and between epilepsy and some forms of manic-depressive insanity.

However, the clinical histories and the course of many mental diseases suggest a more profitable line of inquiry. The facts suggest that we have to do with biochemical processes akin to those of infection and immunity. The history for instance of an attack of melancholia, its gradual invasion, maximum onset of symptoms, gradual subsidence and final recovery, is strangely like that of an infection. That we have to deal with fermentative or enzymotic processes is exceedingly probable. Let us see what the facts indicate.

Intoxications of short duration probably play here a relatively unimportant rôle; in brief intoxications we have to deal with poisonings which the organism successfully resists and, so to speak, disposes of speedily and promptly. For such resistance the organism is admirably equipped. We have in the very beginning the defensive action of the gastric and intestinal juices. If injurious substances notwithstanding gain access to the organism they are submitted to the action of that wonderful laboratory the liver and later to that of other glands and tissues. Substances are oxydized, deoxydized, hydrated, dehydrated or variously combined or disintegrated and finally eliminated. Such is probably the course of intoxications of short duration, but in those of long duration such as are commonly observed in mental disease, the organism doubtless has recourse to the formation of immune bodies; in other words, the organism reacts to intoxications just as it does to infection, namely by a defensive fermentation. The rôle that the fluids of the organism play in its protection we have been taught by Buechner, Bordet, Bouchard, Behring, Roux, Kitasato, Ehrlich and others. The agencies at work have been shown to be substances which bring about immunity by a biochemical action, an action that is probably reducible to physico-chemical terms. In combating the poisons which give rise to nervous and mental disorders, the phagocytes have, it would seem, no opportunity for the exercise of their

special function, but it is not improbable that they join the serum of the blood in furnishing protective substances. The truths revealed in the discovery by Behring and Roux in regard to the curative and preventive action of the serum of animals immunized to diphtheria, in the discovery by Kitasato and Behring of the kindred facts in regard to tetanus and by Simon Flexner in regard to cerebro-spinal meningitis, constitute stories of brilliant human achievement. Ehrlich's results in regard to ricin and abrin and Calmette's in regard to cobra poison are likewise familiar to us all. Nor need we call attention to the specific sera developed against definite microorganisms, the bacteriolytic sera, nor to the sera developed as defensive agents to erythrocytes, the hemolytic sera, nor to the numerous other cytolytic and cytotoxic sera that have been experimentally produced, such as the hepatotoxic, thyreotoxic, neurotoxic, spermatotoxic sera and others that might be mentioned. The doctrine of antigens and anti-bodies is destined it would appear to have an ever widening application. An antigen is, as we all know, a substance which when introduced into the organism is supposed to bring about the formation of a specific antibody. Among such substances are microorganisms, their toxins, cells of various kinds or extracts of their substance, poisonous and non-poisonous proteids and lipid substances.

It would obviously be out of place to dwell here upon the details of the theory of the antigens and antibodies save to recall some of the basic facts, the application of which to our special inquiry will become apparent later. Thus, the body resulting from the union of the antigen and antibody is composed of two basic substances, one the thermostable, the other the thermolabile substance. The thermolabile substance can only combine with an antigen whether the latter be a microorganism, an erythrocyte, a toxin, a proteid or what not, after the latter has been prepared, *i. e.*, treated with a specific thermostable substance. The thermostable substance is the amboceptor of Ehrlich, the fixator of Metschnikoff, the corps sensibilatrice of Buechner and Bordet; the thermolabile substance is the complement of Ehrlich, the cytase of Metschnikoff, the alexin of Buechner and Bordet. It is significant that the thermolabile substance is found in the normal serum.

Let us now turn our attention briefly to the elementary facts

of precipitation, agglutination, opsonin reaction, and complement binding.

If a parenteral injection of an antigen be made into an animal, the serum acquires a new property, *i. e.*, when it is added to an emulsion of the material originally employed there ensues a flocculent precipitate. The antibody which is the cause of the precipitation, is in this instance termed the precipitin. Like other antibodies it has a specific character; that is, it causes a precipitate only with preparations containing the corresponding antigen.

The phenomena of agglutination are briefly summarized as follows: if the serum of an animal immunized to a bacterial culture or its products, be added to a liquid in which these bacteria are suspended, the latter begin to aggregate in masses and to sink to the bottom of the tube; of this the Widal reaction is a familiar instance. The antibodies are here termed agglutins and are of course specific in their action. Very little work has thus far been done upon the phenomena of agglutination in the sera of the insane.

Opsonins are, as is of course well known, substances which prepare microorganisms for consumption by the phagocytes. Opsonins exist preformed in the serum and are probably alexins or complements, *i. e.*, thermolabile substances. In how far they play a rôle in the defense in cases of mental disease of bacterial origin is of course an open question. The opsonic index is on the whole reduced in the chronic insane, though it is increased in some cases of dementia præcox and increased in the larger number of epileptics. It is interesting to know that D'Abundo found the bactericidal properties of the blood increased in paresis, in pellagrous insanity, in mania and melancholia, while in paranoia it was normal or not characteristic.

Complement binding—a reaction which was discovered by Bordet and Gengou—consists in the fact that when an antigen and its corresponding antibody meet, the complement is held fast. This fact can be demonstrated by the addition of a hemolytic system; if the complement has been held fast there is no hemolysis. This is of course the basis of the Wassermann reaction. However, the principle has been applied to the study of other affections than those supposed to be syphilitic. Thus Geiszler immunized rabbits with the serum of various cases of insanity by giving five or six injections into the abdominal region at in-

tervals of five or six days. After the third injection Geiszler observed that the serum of the rabbits immunized with the serum of dementia præcox had acquired the property of complement binding with the serum of all dementia præcox cases, hebephrenics or catatonics, while the same serum yielded no reaction with the serum of healthy persons or that of patients suffering from other forms of mental disease. Similar results were obtained with the precipitin.

When we turn our attention to anaphylaxis, we find that it appears to be increased for tuberculosis in dementia præcox. This points to a connection in this affection between the functional disturbances of the glands of internal secretion and tuberculous infection, a fact to which I called attention several years ago.

The hemolytic action of the sera of the insane is alike interesting and important but cannot detain us here, suffice it to say that they show suggestive and significant departures from the normal in epilepsy, manic-depressive insanity, dementia præcox and alcoholism.

The lymphocytosis of the cerebrospinal fluid, so important as a diagnostic factor, depends upon changes foreign to those thus far considered and need not detain us farther than to point out that it is met with in paresis, tabes, cerebrospinal syphilis, the sclerosis, brain tumors, and in various forms of meningitis.

The rôle of antigens and antibodies, of precipitins and agglutins, the phenomena of complement binding, the play of the opsonins, hemolysins and other substances, suggest that all these processes are the expression of a common biochemical or physico-chemical action, namely, of a fermentation. All of the processes of nutrition, all of the changes exhibited by proteids and carbohydrates, are the expression of such an action and occur under the influence of catalytic and enzymotic processes. Again the fact that ferments consist of two basic substances, first a zymogen and secondly an activator, goes far to prove the identity of fermentation and immunizing processes. Further, just as antigens lead to the formation of antibodies, so do ferments lead to the production of anti-ferments, *e. g.*, pepsin to anti-pepsin, trypsin to anti-trypsin. Again, ferments also manifest the property of specificity; thus each ferment is active upon a specific substrate. Finally, in regard to fermentation in general, the conclusion is justified that it is a universal property of living matter. A point

of practical importance may be added, namely that many ferments preserve their activity for some time after the death of the organism; thus in a measure this subject is open to post-mortem study. The studies thus far made are intensely interesting; *e. g.*, it has been found that antitrypsin is increased in all cases in which the amount of proteolytic ferments is increased and in which a reduction of proteids with a corresponding degeneration of cell elements is taking place. Thus the antitryptic properties of the serum are increased in paresis during periods of increase of symptoms and diminished during remissions. It is interesting also to add that it is diminished after salvarsan administration. Similar phenomena of increase and decrease are noted in alcoholic insanities and on the whole the inference seems warranted that an increase in the antitryptic property of the serum is an unfavorable indication.

Let us now turn our attention briefly to the aspect of the subject of fermentation developed by Abderhalden. The method of study employed depends upon the fact that a foreign proteid substance introduced into the blood excites the production of a ferment, the action of which is to protect the organism against the foreign substance. Such a foreign substance may gain access to the blood from without or from organs and tissues within the body. An instance is the digestion of placental elements by ferments in the blood of the pregnant woman, the blood of the non-pregnant women and of men containing no such ferments. The work which Abderhalden and his pupils have done is already very large in amount and the subject has become so extensive as to be impossible of inclusion in so brief a statement as this address necessitates. Suffice it to say that Fauser and others have tested the digestive or reducing action of the sera of different forms of insanity in relation to the thyroid gland, the sex glands, the brain, the kidneys and other organs. Many remarkable observations have been made.

The time at my disposal will only permit of a few additional observations; these will be somewhat in the nature of general statements. First, proteids and lipoids alone can evoke the formation of antibodies, and in this process lipoids appear to play the rôle of activators, and their injection seems to bring about an increased formation of immune bodies. Second, the specificity of immunity and fermentation processes does not appear to be

entirely absolute. It is very probable that such biological conceptions as antigens and antibodies will be replaced, indeed, are being replaced, by more precise chemical conceptions. It appears that the curative action of a given serum depends upon its containing a larger amount of substances already normally present in the body. Syphilitic antigens, for instance, may be derived from various normal tissues but under the influence of the spirochete they increase in amount. Third, it is exceedingly probable that the physical structures of colloids is able to explain the processes of fermentation. Colloidal substances seem especially able to evoke the formation of antibodies. Fourth, in the struggle with intoxication and auto-intoxication, a most important rôle must be assigned to the glands of internal secretion. The various other tissues and organs of the body, in addition to their special functions, possess also the function of internal secretion inasmuch as they elaborate, as a result of their functional activity, and pass into the blood and other liquids of the body, certain substances which in one way or another act upon other organs and thus add to the number of the hormones as we now term them. The life of the organism is that of a well coördinated chemical whole. It is the disturbance of this coördinated chemical whole either by the introduction of foreign substances from without or by the breeding of foreign substances within the body through the breaking down of the coördination, that causes intoxication. Disturbances of metabolism, using this term in its widest sense, are therefore the basic factors in mental diseases, and this truth brings mental diseases properly within the province of internal medicine; but in order that they should be viewed in their proper perspective, the following important facts must be borne in mind. Mental diseases, as just indicated, separate themselves into two great groups; first one in which the disease is due to infection or exogenous poisoning and second one in which the disease is due to endogenous poisoning.

In regard to the first group, our therapeutics, our ability to interfere to the advantage of the patient, is in a far better position. In the second group, we have disorders which occur in organisms whose structure is chemically and physically essentially defective, deviate and aberrant. Their peculiarities are unfortunately hereditary and innate. These peculiarities do not manifest themselves markedly as a rule until the period of life is

reached at which metabolism assumes a special activity, that is, at puberty or later. At puberty, in adolescence, in early adult life, the sexual glands and with them all of the other glands of internal secretion and hormone producing structures and tissues, take on a new rôle, enter upon a new chemistry, and if the organism be badly constructed, be badly put together, this chemistry becomes aberrant and toxic, and mental disease results. How powerless we must necessarily be in the face of such facts need not be dwelt upon. However, two hopes are held out to us; first, in the manic-depressive group in which the entire picture is one of the predominance of antigens in the early stage—in an attack of melancholia or mania as the case may be—and of the predominance of antibodies in the later stage, it is by no means impossible that sera will be devised which will enable us to cut short such attacks and perhaps to practice preventive immunization for recurrences. It is with regard to the heboid-paranoid group, the great group of dementia præcox and paranoia, that the outlook is the most discouraging. Here we must necessarily be limited to simple physiological therapeutics, but even here if these procedures be properly applied much can in given instances be accomplished. It is, however, in the field of eugenics that the second hope arises, forlorn and distant as this may be. We physicians, however, know that defects of stock arise largely from the infections and poisonings from which the ancestors have suffered. Syphilis, tuberculosis, alcohol, are the potent causes of structural and chemical deviation and degeneration, much more potent than insufficient food or exposure. It is in the minimizing of these factors that the hope of the future lies.

THE MORPHOLOGY AND FUNCTIONS OF THE CORPUS STRIATUM

BY JAMES HENDRIE LLOYD, M.D.

It is my purpose this evening to discuss the corpus striatum, first with reference to its morphology, second with reference to its functions and diseases. The paper, I fear, will not be an orthodox one; nevertheless, I have no apologies to make for it beforehand. As in the case of every man who assumes a rebellious attitude towards recognized authority, I may meet with strong disapproval, but as one of the few remaining original members of this society, I will try on this its thirtieth anniversary to exemplify the spirit in which we have worked together for thirty years, and which has given all of us the right to play the part of free lances whenever we have felt inspired to do so.

The corpus striatum has loomed very large recently in pathology. To be exact I ought perhaps to say the lenticular nucleus; but as the lenticular nucleus is only one of the broken fragments of the original striate body, it serves my purpose better to adhere closely to morphology, and to include the part in the whole. The striate body is the organ under review.

The comparative morphology of the corpus striatum is one of the most interesting studies in neurology. It is a study that carries us far back, even to a period before the origin of the vertebrates. There is no part of the brain that has such an instructive story to tell, if only it is looked at knowingly. Before the amphioxus was born, or even the larval ammocoetes of the earliest vertebrate was brought forth, the corpus striatum had begun to do duty as a brain mass in the arthropods. It was in fact the whole brain in the arthropods, being known to-day as the supra-esophageal ganglion, and it continued to do duty as practically the whole fore-brain in the earliest vertebrates up to and including the fishes.

This subject has long interested me for its bearing on the physiology and pathology of the brain; and the interest has now

become acute because of the enormous claims put forth by some neurologists for the part alleged to be played in disease by the lenticula.

I am disposed first to enter a gentle protest against what seems to me to be a cloisteral method of manufacturing syndromes; and to call the attention of this society to a mode of view which in my opinion has too much been neglected. If we were sometimes to take a larger survey of the nervous system, in its evolution from lower forms, we might acquire a wholesome distaste for the artificial point of view which is apt to be too much in favor with us neurologists. At any rate, it is refreshing and invigorating at times to go far afield. I invite your attention, therefore, to a brief excursus into the remote past.

I shall base my discussion of the morphology of the corpus striatum largely on the work of Gaskell,¹ for I am not an original worker in this field. In his treatise on the origin of the vertebrates Gaskell undertakes to establish the thesis that the vertebrates were developed from the arthropods, and that the cerebrospinal axis of the vertebrates was originally developed around and including the gastrointestinal tract of this lower form. If you will stop to reflect a moment, you will recall that in the arthropods, which include the crabs and the spiders, as well as in still lower forms, such as the insects and the segmental worms, the nervous system lies ventrad of the gastrointestinal tract. It consists of a chain of ganglia lying beneath the creature's ventral surface; whereas in the vertebrates just the opposite is true, for in them the cerebrospinal system is located dorsad of the stomach and intestine. For a long time this was a stumbling block to evolutionists, for the problem stared them constantly in the face how the vertebrates ever succeeded in getting their nervous system on top of their bellies. The evolutionists resorted to all sorts of expedients to explain this apparently inexplicable thing; and such an eminent morphologist as Owen even proclaimed his belief that before the arthropod could have developed into a vertebrate, the creature must have turned over on its back. Of all the somersaults on record this was entitled to the prize!

But Gaskell sees no need of resorting to such evolutionary tactics. For him the arthropod did not need to turn over on its

¹ The Origin of Vertebrates, London, 1908.

FIG. 1. In the arthropod is seen the large cephalic stomach, entered by the esophagus, which opens on the ventral surface. In front of this opening is a large ganglionic mass, the supra-esophageal ganglion. Behind the opening is another large mass, the infra-esophageal ganglion. These two ganglia are connected by nerve tracts which embrace the esophagus, and which represent in a rudimentary form the future cerebral peduncles. Beneath the gut is the chain of nerve-ganglia. The elements of the future vertebrate cerebrospinal axis, here represented, are as follows: the supra-esophageal ganglion is to become the primitive fore-brain mass, or corpus striatum; the infra-esophageal ganglion is to become the ponto-bulbar region, or hind-brain; the esophagus is to remain as the infundibulum; the chain of ganglia is the future spinal cord; the membranous wall of the cephalic stomach is the basic membrane on which is to be built up the cerebral hemispheres, especially the anterior portion of this wall, which is to be called the pallium.

FIG. 2. This represents the ammocetes, a larval form of one of the earliest vertebrates. The advance made can be seen at a glance. The gastrointestinal tube has now become a cerebrospinal canal. It no longer opens by an esophagus, but the place of its former opening is indicated by infundibular structures. Well in front lies a large ganglionic mass on the floor of what we may now call the primary fore-brain vesicle. This mass, formerly the supra-esophageal ganglion of the arthropod, has now become the corpus striatum. In this early form it represents pretty much all that there is of a fore-brain, for this animal, as can be seen, had no cerebral hemispheres. Behind and on the floor of the canal are the hind-brain structures, the former infra-esophageal ganglion; and above is the pineal eye. Between this pineal eye and the corpus striatum is only a thin membranous wall, without nerve tissue. This is the pallium, upon which is to be built the future cerebral hemispheres.

FIG. 3. In the fish the corpus striatum is still the real fore-brain, for the pallium is still undeveloped, and is nothing as yet but a membranous wall. Connected with the corpus striatum, but not shown in the figure, is an immense rhinencephalon, or pair of olfactory lobes, extending far out in front. In fact, the olfactory and optic nerves are from the very beginning in the arthropods connected with, or have their origin in, the fore-brain structures; and this continues to be so in the highest vertebrates. The fishes have also developed large optic lobes and behind them a cerebellum. The infundibulum, with the contiguous pituitary, is plainly seen.

FIG. 4. The amphibia show a beginning cerebral hemisphere; and this, it is to be noted, has developed on the pallium, or anterior membranous wall. It contains a few ganglion cells. We now begin to see something that looks like a ventricle, which extends down into the infundibulum. What is left of the pallium has become folded inwards as a velum interpositum and choroid plexus. The corpus striatum is still the better part of the fore brain.

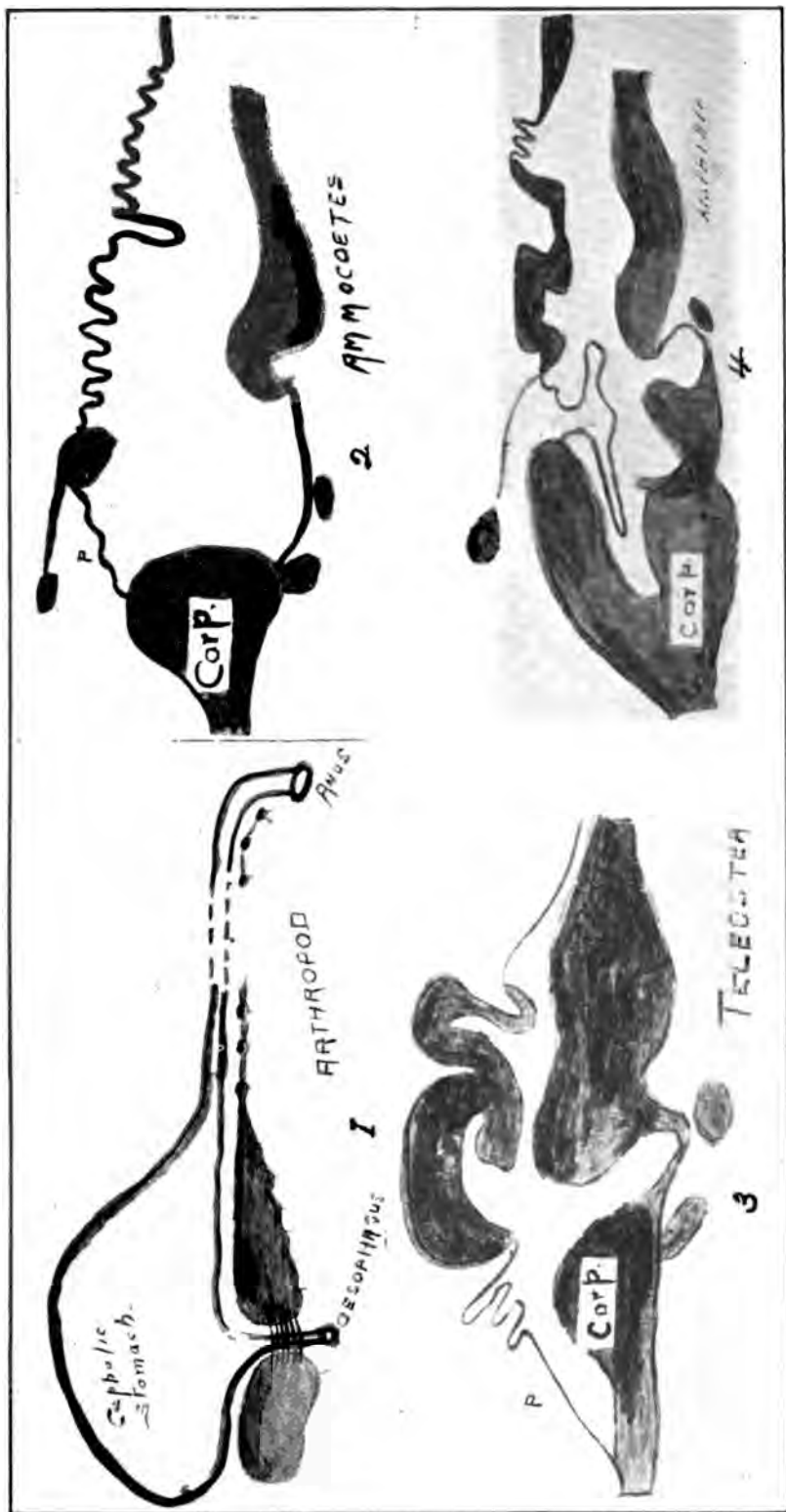
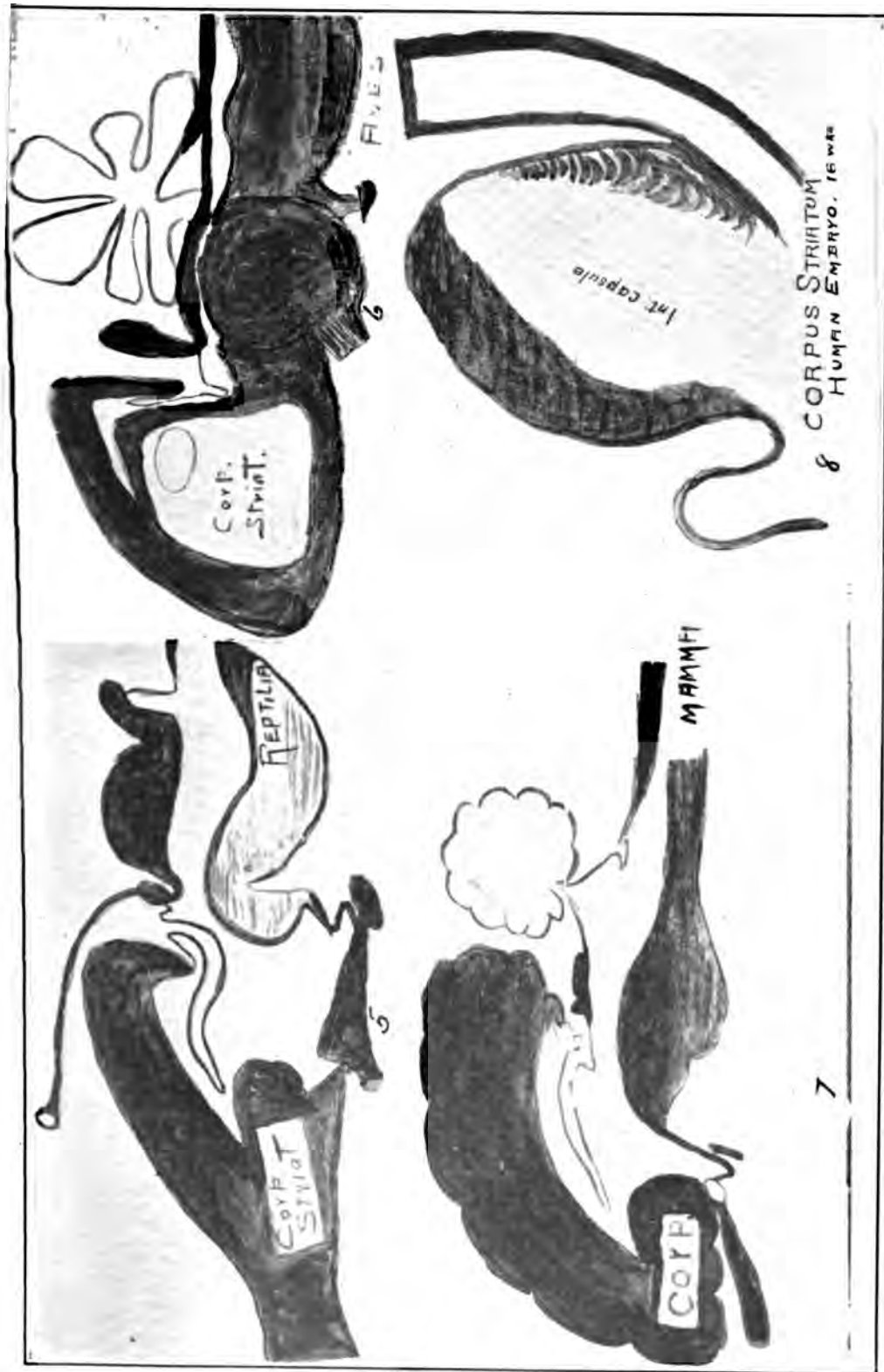


FIG. 5. In the reptilia the chief thing to be noted is the great increase in the size of the cerebrum, growing on what was the pallium. We first now, in fact, begin to have a true brain in our modern conception of a brain, for, according to Edinger, we first meet in reptiles with a deposit here of pyramidal cells, disposed in several layers and covering most of the surface—a true brain-cortex. The corpus striatum is beginning to be buried beneath this rapidly evolving pallium. Moreover, as it was necessary for this growing brain-cortex to extend its connections to the pontobulbar structures and the spinal cord below, a way was established by taking the shortest cut, and this shortest cut was right through the corpus striatum. In fact, the corpus striatum was literally broken into two fragments; and it remains so to this day. We call these fragments the lenticular nucleus and the caudate nucleus. That short cut is the internal capsule.

FIG. 6. The bird's brain is remarkable for the great development of the cerebellum; and for a corpus striatum that attains a relative size and complexity found in no other class of animals, while the formation of the cortex does not much increase. It would seem that the bird must still have a very active corpus striatum, as this structure forms the larger part of its fore-brain.

FIG. 7. When we come to the mammalian brain, which is the highest type, we find a fully developed cerebrum. This cerebrum, growing on the pallium, extends farther and farther backwards, gradually in successive forms covering more and more of the mid- and hind-brain structures, until in man its enormous mass overlies even the cerebellum. Here is the true brain, which has gradually dispossessed the little primary corpus striatum and buried it out of sight. It is an instance of the evolutionary triumph of the greater over the less.

FIG. 8. The fact that the corpus striatum has no direct connection with the brain-cortex, is shown in this figure after Edinger. It is seen that the pallium, or brain-cortex, incloses the striatum much as the skin of an orange incloses the pulp; but there is no direct connection. This can be demonstrated by careful dissection at the line of contact between the two at the external capsule. They can be separated without tearing. Moreover, it is evident from this demonstration that nature in her evolutionary process has not spared the corpus striatum, for she has literally broken it into two fragments in her need to force a way through it for the motor and sensory tracts in the internal capsule.



back in order to become a vertebrate. The process was much simpler. In brief, Gaskell holds that the nervous system of the vertebrate was evolved by a process of building up nervous tissue around the old gastrointestinal tube of the arthropod. To use his expression, the gastrointestinal tube was gradually invaded by nerve-tissue. The infundibulum thus represents the old esophagus; the pineal gland is the old median eye; the corpus striatum is the supra-esophageal ganglion; the ventricles of the brain are what remains of the arthropod's stomach; the spinal canal is what remains of its gút (still opening in the human embryo into the anus); and the cerebral hemispheres are masses of nerve-tissue gradually built up on the anterior membranous wall of the cephalic stomach, the so-called pallium.

A survey of the accompanying figures will explain this thing in a diagrammatic way. These figures are taken from the works of Gaskell and of Edinger,² and have been redrawn by Dr. Ludlum. They represent the evolution of the nervous system from the arthropods to the highest vertebrates.

From all this the evidence is clear to me that the corpus striatum has gradually sunk to a position of infinitesimal importance. It is doubtful, in fact, whether it continues in the human brain to exercise any functions whatever. If we choose to regard it as a vestigial organ, we can find plenty of analogies for such vestigial tissue in the mammalian brain. As already said, the pineal gland is a vestigial eye, but in spite of the fact that it ceased long ages ago to exercise the functions of an eye, it still presents some evidence of retinal tissue in its make up. If Gaskell is correct, the infundibulum is the old esophagus, but in spite of the fact that no food has passed through it for untold æons, it still retains an intimate connection with the pituitary body; a body which is in no sense a part of the brain, for it contains no brain tissue, but probably represents the coxal glands, which in the arthropods were located near the mouth opening and may have had some important function in excretion or nutrition (a fact which throws a strong light on what we know now of the pituitary as an organ of internal secretion, influencing in an occult way nutrition and development). The rhinencephalon, or the brain of smell, has undergone an enormous de-

² Structure of the Central Nervous System, Philadelphia, 1890.

generation in the human brain, as anyone can see who compares it with the same organ in the carnivora, or even in the horse or ox.³ Most remarkable of all, the whole cerebrospinal canal has completely abrogated its original place in nature as a gastrointestinal tube.

If any one contends that such a metamorphosis is inconceivable, his attention need only be called to what took place in the evolution of a lung-breathing animal from a fish, which breathed in the water by means of gills or branchiæ; and if he wants an example of what nature can do by way of perpetuating organs which during millions of years have become absolutely useless, he need only look at the gill-slits in the human embryo. Surely if nature can preserve the gill-slits of a fish in the neck of a human embryo, she can also preserve the useless remnants of the fish's brain in the broken fragments of the human corpus striatum.

So far from such astonishing metamorphoses being the exception in evolution they are rather the rule. As Johnston says,^{3a} both the rise of new structures and the passing away of old ones have brought about shifting of organs which is of great importance in determining the structure of the nervous system. This shifting of organs, and transference of function from one organ to another, has been largely the method by which evolution has proceeded in what is called cephalization or development of the head and brain in vertebrates.⁴

If now we resort to histology, we are helped but little to an understanding of this subject. In fact, we are rather confused. The histology of the corpus striatum is a hopeless maze. No man has ever made much out of it yet, so far as I have been able to ascertain. And no man will ever settle this broad question by

³ Sisson, *Anatomy of the Domestic Animals*, pp. 837, 844, and 848.

^{3a} *Nervous System of Vertebrates*, p. 69.

⁴ Gaskell's theory of the origin of the vertebrates has been criticized and rejected by some embryologists because it contravenes the principle of the three primary layers, the epiblast, mesoblast, and hypoblast. His theory, it is argued, makes the impossible claim that what was hypoblast in the arthropod has become epiblast in the vertebrate. Gaskell confutes this criticism, but the argument is too long and technical for me to attempt to reproduce it here. It can be read by anyone interested, in chapter XIV of his book. He says by way of conclusion: "Of recent years grave doubts have been thrown upon the validity of this theory (*i. e.*, of the three primary layers) . . . So much is it now discredited that any criticism against my theory, which is based upon it, weighs nothing in the balance against the positive evidence of recapitulation already stated," p. 486.

peering through a microscope. Van Gehuchten says that we are ignorant of the morphological connections of the basal ganglia, just as we are ignorant of their functions. Wernicke believed that the gray matter of the caudate nucleus and of the external segment, or putamen, of the lenticular nucleus gave origin to nerve fibers analogous to those which come from the cortex of the cerebrum. This might merely indicate, if Wernicke is right, that the corpus striatum still preserves vestigial tissue which is a reminder of the functions it once preformed as a primitive brain mass. It need not indicate that these fibers retain any of their primitive importance. Cajal says that fibers from the cerebral cortex pass through, but not to, the lenticula, to be distributed to inferior parts of the cerebrospinal axis; in other words, they have nothing to do with the lenticula. Edinger points out that some fibers connect the caudate nucleus and the lenticular nucleus by passing through and across the internal capsule. Such fibers may merely indicate the original connection between these two fragments of the corpus striatum, and may have little if any functional importance. There is no evidence of any direct connection between the brain cortex and the corpus striatum. Edinger describes fibers from the brain cortex which pass through the globus pallidus and are gathered up in the ansa lenticularis beneath the lenticula—in other words they merely pass through the lenticula. Histology has its place, but conclusions drawn from it as to function may be most misleading. I have already illustrated this fact when speaking of the pineal eye, which certainly has not seen the light for untold ages.

Another fact, of great significance, is shown by Johnston⁵ in the histology of the striate body in the fish; namely, that the striate body of the fish is closely connected with the olfactory brain or rhinencephalon. There is nothing like this found, so far as I know, in the human corpus striatum. It has apparently long since lost any such connection.

When we turn to experiments for light upon the functions of the striate body, we are not much better off. These experiments have usually been performed by electrical excitation. Ferrier,⁶ who reviewed this whole subject, including his own experiments

⁵ *Op. cit.*, p. 299.

⁶ *Functions of the Brain*, pp. 409, 410.

as well as those of others, inclines evidently to the belief that the effects produced by irritating the basal ganglia are due, not to the irritation of the ganglia themselves, but to the irritation radiating to the fibers of the internal capsule. He holds that, provided the cortical motor centers and their pyramidal tracts in the internal capsule remain intact, the ganglionic masses of the corpus striatum may be destroyed, or at least extensively injured, without giving rise to appreciable symptoms. This puts their functions far below par. Ferrier combats Bechterew's fanciful idea that there is somewhere in these basal ganglia an emotional center—an idea of Bechterew's which has been much exploited to explain the automatic laughter of pseudo-bulbar palsy. But Ferrier apparently believes that there is a difference between the higher and lower animals, and that in the rabbit, for instance, the corpus striatum may still perform some functions. This supports the idea that it is a deteriorating organ, for the rabbit is low in the scale of mammalian animals, and may still have a corpus striatum that is worth something to him.

The most recent experimental work on this subject is that which has been done by Kinnier Wilson—a piece of work which is praiseworthy, and is also significant because of Wilson's prior article on diseases of the lenticula.⁷ He is able apparently, as a result of his experiments, to claim no more for the corpus striatum than that it exerts a "steadying" effect upon the motor neurons in the internal capsule. This is the only comfort he can get in support of his peculiar views about the pathology of the lenticula. But far more important, Wilson concludes that "there is no vestige of evidence that in man the corpus striatum is capable of assuming a long-lost function and of acting vicariously for the motor pallium. Its true motor function has gone never to return." He further says that when the "superman" is finally evolved, he will probably have no corpus striatum.

By way of a mild criticism of Wilson's conclusions I should like to ask what is meant by a "steadying" or "toning" effect? The term is novel to me. Is it possible that one neuron needs another neuron to stand alongside of it and keep it toned up? If that is so, what keeps the toner toned up? Such a conclusion seems like an endless chain.

⁷ Experimental Research into the Anatomy and Physiology of the Corpus Striatum. Brain, Parts iii and iv, 1914.

From this review of the morphology of the corpus striatum I am led to the opinion that the striate body in the human brain is a vestigial organ, representing the original fore-brain mass in the earliest ancestral types of the vertebrates. It is a little played-out fishes' brain, a submerged brain, broken into two fragments, lying deep in the cerebral hemispheres, which have been developed over it in the original pallium, or membranous wall of the fore-brain vesicle, and have completely usurped its functions and added immensely to them. The difference between the two is the difference between the human brain cortex, on the one hand, and the supra-esophageal ganglion of a crab or a spider, on the other hand. They lie at the extreme poles of the evolution of the nervous system.

The bearing of this demonstration on pathology remains to be discussed—but time will only allow me to make a brief statement of my views in a few general propositions. I have gone over a series of 10 or 12 brains in my own collection at Blockley, but I do not intend to bore you with case reports.

First, I hold broadly that the symptoms caused by lesions of the lenticula are the symptoms merely of various affections of the fibers of the internal capsule. There is no such thing as a lenticular syndrome properly so-called.

Second, the lenticula derives its sole importance in these cases from its contiguity to the internal capsule, and from the blood vessels which run into and through it—vessels which are very prone to be the seats of hemorrhage or obstruction.

Third, when such lesions occur on both sides (that is, when both lenticulæ are involved), especially in progressive or successive attacks, there is produced the condition known as pseudo-bulbar palsy, which is properly a bilateral capsular syndrome. Nothing is to be gained by renaming it "progressive lenticular disease," because the mere renaming of a thing does not add a whit to our knowledge of it.

Fourth, pseudo-bulbar palsy has many varieties, and not a few aliases, but its main features are dysarthria, dysphagia, automatic emotionalism, and various affections of the motor paths to the upper and lower limbs. These motor paths may be partially or almost completely paralyzed, or only paretic, and the limbs may exhibit various motor disorders, such as tremor, spasticity, atetosis and even incoördination. These varieties depend upon the

degree of involvement of the motor fibers in the internal capsule, whether they are totally paralyzed, partially paralyzed, or irritated and in other ways disordered in their functions and nutrition by a disordered blood supply. There is no proof that one set of fibers is needed to keep another set of fibers in tone: it is more probable that a deranged blood supply accounts for the various freaky symptoms, which are seen in these cases. The reflexes may, or may not, be affected, according to the degree of involvement of the pyramidal tracts.

Fifth, dysarthria in these cases is caused by involvement of the motor tracts in the capsule running from the cortical speech centers above to the ponto-bulbar nuclei below. The facio-oro-lingual center in the brain cortex is probably located near the foot of the ascending frontal gyre, but the exact position of its descending tracts in the internal capsule is not so definitely known; they are probably placed well forward in front of the knee. The claim that the striate body may be a coördinating center for speech is to my mind entirely fanciful, and is discredited by the morphology and phylogenetic place of that body. It is inconceivable that the fish's brain could have any speech function. Speech is a function of the human brain cortex alone—one of its highest specialized functions. To place any part of it in the striatum is to go counter to all the indications of morphology. It is only the descending tracts from the speech centers in the cortex that are involved in these cases.

Sixth, the mechanism of automatic laughter and weeping is not readily explained, but to attempt to explain it by arbitrarily locating an emotional center in the basal ganglia is, in view of what we have just seen, positively grotesque. Emotion is a form of ideation: it is a cortical function. I grant that if a fish ever laughed, it must have laughed with its corpus striatum; it had nothing else to laugh with; but I do not believe that a man laughs in that way. I have attempted elsewhere to explain this automatic emotionalism by suggesting that it is strictly analogous to other automatic phenomena, as, for instance, the action of a decapitated frog in wiping off a drop of acid from the skin of one leg by using the other foot; or the action of the decapitated rattlesnake, which, if its tail is trodden on, will raise its headless trunk and strike. The various nuclei of the many muscles concerned in the act of laughter have become trained to a coördinated ac-

tion. They may go off, as it were, by themselves when they are cut off from the controlling centers in the brain cortex; and this condition is well presented by lenticular lesions involving the capsule, when such lesions are bilateral.

In conclusion: as I said at the beginning, I have gone far afield; and I realize that I may not very successfully have condensed in a paper of fifteen minutes what it has taken nature whole geological epochs to accomplish. The anniversary of our beloved society put me in a reminiscent mood, and when I got to thinking of the past I did not stop until I reached those halcyon far-off days when the infundibulum was the old esophagus.

THE PSYCHOLOGY OF MISERS

BY CHARLES W. BURR, M.D.

PROFESSOR OF MENTAL DISEASES, UNIVERSITY OF PENNSYLVANIA,
PHILADELPHIA, PA.

The student searching for data to serve as a basis for a classification of men according to mental type, who believes that true insanity is developmental, the symptoms varying with the personality, and that if we had a psychological classification of normal man it would aid in classifying the abnormal, who sets for his task, or rather his pleasure, the reading of lives in expectation of finding scientifically important and interesting matters recorded, and who is trying to discover why individual men and women are what they were and not otherwise, what factors in heredity make them, what elements in environment mould, when they do mould, the immortal, ancestral protoplasm, soon becomes vexed with biographers and autobiographers. Some of the latter, it is true, reveal quite unwittingly traits of character, of which they themselves are entirely unconscious. Thus Herbert Spencer's ponderous autobiographic tomes show him to have been not the modest student he thought himself but one of the world's greatest egotists. Many autobiographers evince a complete faith in their own power, accepting unconsciously, for they are often far from religious and unlearned in scholastic lore, the theological dogma of free will and believing they made themselves and determined their careers, whereas some psychologists at least, even in this day which is trying to persuade itself to believe that all men are mentally and morally equal if only they be given external advantages, especially schooling, think we are what we are because of inherent qualities in the parental germ cell and sperm cell. For the most part books about people are so loaded down with trifling matters of external life, with things that throw no light on the development of the man and his personality, that the psychologist gets but little from them. Further, I must confess that in certain regards we owe more of our knowledge of the

real nature of man, and after all this is psychology, to the artists, the great poets, novelists and dramatists, than to the technically educated students of science. I mean of course from the point of view of what one may call clinical, personal, descriptive, psychology, and not as regards the formulation of scientific theories as to mental development and action. I mean the difference between Shakespeare and some of the men who are reading psychology as a major in a university course leading to the Ph.D. degree. The reason is that the great artist like the great man of science, and there is much more resemblance between the two than meets the common eye, sees what he looks at, sees things as they really are. The poet, the mathematician, and the real discoverer in the natural sciences are alike in that all have tremendous power of normal imagination while scientific students of lower rank, those who become students by accident and do not, to use a theologic term, have "a call," see only what is pointed out to them and lack imagination entirely just as the little poet may appropriate the mechanics of a master's verse but can not supply a soul. Thus Harrison Ainsworth's novel, "The Life of Jack Sheppard," mere melodrama as it is and its author far from being a genius, gives a truer picture of the real criminal, what his emotions are, what things control his conduct, what his view of life is, than some learned treatises written by self-styled experts in criminology which we are compelled to read as part of the day's work.

In recent years the clinical psychologic study of the dead has become quite popular but usually the problem has been attacked from the wrong end. Instead of studying a large number of men of a given type without bias and prejudice and then drawing a conclusion, the common plan is for an author to assume the truth of a theory and then to search through literature for evidence in support of it. Because Shakespeare's signature to his will is tremulous, therefore, say these writers, he had paralysis agitans, therefore he was a degenerate, therefore all genius is degeneracy; Goethe had an hallucination in early life, therefore geniuses are all hallucinatory. Examples could be multiplied indefinitely. Most of the psychological study of the dead has been in reference to genius, an unconscious tribute to the fact that the world owes all progress to unusual men, to the real leaders, and nothing to the mass, to the common people, whom

it is an acquired characteristic of today to endow with all the virtues, including wisdom and clear thinking. The real science of personal psychology, the classification of men, remains almost unknown because so little studied.

The above is preliminary to the statement that in this little study of one particular type of man I have found it difficult, often impossible, to get the facts, *i. e.*, the kind of facts which throw real light upon and explain why any given man is the man he is. My information was of necessity gotten from sources where one does not look for accuracy of statement. All the books about queer people, eccentrics, misers, murderers, monsters, and the like are written solely with a view to entertain, not to instruct, and misers belong to a class about whom serious biographies, poor for our purpose as biographies usually are, are not written.

Let me relate to you briefly the lives of a few misers and then see whether we can draw any general conclusion. Time and space are so limited that I can do no more than catalogue qualities and give incidents which indicate character. In any such study the Dancer family naturally comes first, because they have become immortalized in serious literature and because in them miserliness extended over three generations (a very rare instance of direct heredity of pathological psychological personality) though Daniel was the most notorious, or famous, or infamous; you must choose for yourselves individually which is the most appropriate epithet. The grandfather, father and all the brothers and sisters of Daniel Dancer's generation were alike misers. Of the oldest generation little is known except that they were landholders and ought to have been people of such character as to have been held in esteem and to have had positions of responsibility and respect in their community. They were held in contempt. Daniel was born near Harrow in England in 1716 and was the oldest child. His avarice, it is said, appeared only after the death of his father, from whom he inherited a comfortable fortune. He believed that the old gentleman had concealed more than fifteen hundred pounds in the house and was afraid his brothers would find it and not give it to him, the heir, and consequently kept very quiet for some time about his suspicions, but two years later on removing an old gate, about two hundred pounds in gold and bank notes was found between two

pewter dishes buried under one of the gate posts. The remainder of the hidden money, if there was any, was never found. He lived with his sister, whose nature was like his own. He is particularly interesting to physicians because of his poor opinion of us. Once, if not many times, when his sister was ill he said "all the gentlemen of the faculty are medical tinkers, who, in endeavoring to patch up one blemish in the human form, never fail to make ten." Once he found in the field, or by the road, a dead sheep which he took home and he and his sister made it into meat pies which he said tasted very good. When his sister died he hired another miser, a man much like himself, to be his man and together they used to go out hunting for bones wherewith to make soup. His rooms were never cleaned. He would walk two miles rather than not assist in manuring his own lands and even picked up dung on the common. Curiously enough he had a pet dog, which was fed a pint of milk daily but when the dog was found to be a sheep thief his master took him to the village blacksmith and had his teeth broken off. None of the writers give his reason for doing this; whether he acted in anger or to punish the animal. He never had a light in his house save a candle to see him to bed. Once burglars broke into his house but got little; afterward, however, he sent much of his gold to a safer storage place. Once Lady Tempest, who was always kind to him, sent him a trout stewed in claret. It congealed from the cold and in order to warm it, lest eaten cold it should make his decayed teeth ache, he took it to bed with him and so warmed it. His house was a miserable building, but after his death money was found scattered everywhere. Notwithstanding his extreme avarice he was never dishonest, but absolutely straight in all his financial transactions. He seemed to want gold for the mere pleasure of hiding it, fondling it, playing with it and he would rob one pot to enrich another. He lived all his life in his parental home, but allowed the lands about the house to grow into a veritable wilderness. He seems to have worn one coat for many years and wrapped his legs in straw bands. After his sister's death he had a law suit with his brothers about the inheritance and recovered from her estate 1,040 pounds as the price of board for thirty years and one hundred pounds extra for the last two years, during which he declared she did nothing but eat and lie in bed. He died at seventy-eight years.

William Jennings was born in England in 1701. His father, a very wealthy man, died while having built a magnificent country seat and left the son two hundred thousand pounds. The son stopped all work on the house and dwelt in the basement, the floor of which was some ten feet below the surface of the court. It was divided into three rooms, the furnishing of which cost about twenty pounds. He showed some remnant of family pride in having his meals served in this dismal place on the family plate. His peculiar attitude toward expense was shown by the fact that the food left after a meal was never permitted to be served again nor was it given to the poor but express orders were given that it should be fed to the dogs. He was a constant attendant at the fashionable gambling hells of London, not, however, in order to play but to lend money to the unlucky gamblers, his interest charge being a shilling in the pound daily and he purchased a house in London so that he could be nearer at hand to carry on his money-lending business. He was in no sense a social man, but attracted to him some few people by promising to remember them in his will. He did write a will leaving the bequests but he never executed it and after his death his estate, a little less than a million, came near getting into the chancery court, as two noble families each made claim to it. The matter was compromised. It was said of him after his death that he was never known to do one single charitable act. He showed the real character of the miser in that he kept large sums of money unemployed. It was estimated that for twenty years before his death he lost two thousand pounds yearly by idle money. In one of his houses he always kept twenty thousand pounds in cash and in the other a still larger amount. He died in 1797 in the ninety-seventh year of his age.

James Taylor was born in Leicestershire, started life as a weaver and later became a stock broker, in which business he amassed two hundred thousand pounds. His raiment was ragged, his food indifferent and scanty, and his bed was rags and straw on the bare floor in a house which scarcely protected him from wind and storm. Once he invited his friend Daniel Dancer to dine with him and two banker's clerks to take part in the feast. The acolytes of finance found him on their arrival boiling a single mutton chop in a sea of water to make soup for the feast. While he was out of the room they threw some candle ends in

the pot. The mess was eaten by the two queer cronies, but meeting the clerks later Dancer had them arrested for stealing his candles. History relates, probably as accurately as history usually does, that the men cleared themselves by proving he had eaten the candles and as they had become a part of himself he had not been deprived of them but had simply put them to an unusual use. He always bought a twopenny steak, in the market, a sorry piece, an outside piece, grown black by the wind, fly blown and odorous. He use to say "meat was nothing, unless it smelt as well as tasted." He even drove a hard bargain with the church for the salvation of his soul. He was ill, and fearing death, sent for the proper church officials. He paid them twelve hundred pounds for prayers for the rest of his soul but made them return him a year's interest by way of discount for cash payment. His name therefore appears, or did as late as 1813, on the donation board of the church of St. Saviour in London. He died in 1793.

Edward Nokes was a tinker till six weeks before his death, at the age of 56, at Hornchurch in Essex. Almost the only human attribute he had was his fondness for spirits, of which he drank nearly a quart daily without, however, ever showing signs of alcoholic merriment. He fed himself, his wife, and children on offal and washed his shirt in urine. When any of his children died he had a deal box made and carried it on his shoulder to the place of burial and returned home careless and unconcerned. Shortly before his death he gave orders that his own coffin should have no nails in it and that no mourning should be worn at his funeral. On the contrary the pallbearers and undertaker were garbed in striking fashion. He kept large sums of money in a bowl hid in a brick kitchen. Though his surroundings at death indicated abject poverty he really left between five and six thousand pounds which his long-suffering wife, in the absence of a will, inherited.

One is tempted to believe that the next of my heroes is mythical or at least the victim of imaginative and lying tongues, so strange and varied is the history recorded in the books. I have not been able, in the time at my disposal, to trace his history to its original source and give it to you for what it is worth, assuming no responsibility for its accuracy. John Owen lived in London before there was any bridge over the Thames. He

rented for many years the right to ferry people from Southwark to the city by boats. Though he became very rich he lived as though in poverty. He had a daughter, pious and beautiful (a heroine can not be ugly, though nowadays she may be far from pious), and on her he spent money lavishly for education, but when she grew to womanhood would suffer no man near her. However, the inevitable happened: a young man appeared and made quick love to the heroine while the father was gathering fares on the ferry. Meanwhile things went on, in the place the girl called home, as usual. The father warmed the family black pudding in his bosom while rowing and gave it to his family and servants therefrom. He searched the dung hills at night for bones to make soup. He ate food his dog refused (dried bits of mouldy bread). Once, and the thing ended in a tragedy, he feigned sickness and death to save two days' food, thinking that while he lay dead his servants would not be so unnatural as to eat. He told his daughter of the trick and she, though unwillingly, consented to take part. He was laid out for dead, wrapped in a sheet with one candle at his head, another at his feet, but his apprentices so far from being saddened by his death were overjoyed. They skipped, and played and ran as they had never done before, ate all the food in the house and sent out for beer and other luxuries. The old man, dumb with amazement and vexation at the conduct of his servants and at the money loss from such feasting, rose wrapped in the death sheet and taking a candle in each hand stalked into the adjoining room intending to rout the merrymakers out for their boldness, but one of them thinking he was in very truth the devil come to the world to welcome his future guest, struck out his brains with the butt end of a broken oar. The daughter's lover hearing of the death started for town in such haste that his horse threw him just as he was entering London and he broke his neck. The girl became bereft of her senses as a consequence of the double tragedy. The father had been excommunicated on account of the manner of his life and was denied Christian burial, but the daughter bribed the monks of Bermondsey Abbey, in the absence of the abbot, to get him buried. The abbot on his return home discovered what had happened and had the body disinterred, put upon the back of an ass and asked God to take it where it deserved to be buried. The ass, evidently a very knowing beast, went

unguided to an execution place, dumped the body under the gallows and went on his way quiet and undisturbed. A grave was instantly made and the body tumbled in and covered with earth. The daughter, being troubled by a multitude of suitors for her hand and fortune, retired to a nunnery and gave her wealth to the church.

Mr. Ostervald, a French banker, though leaving 125,000 pounds, died of want in Paris in 1790. A few days before his death he refused to buy a little meat to make soup saying, "I would like the soup but I have no appetite for the meat, and what would become of it?" At the very moment he had thousands in bank notes hidden in a silken bag attached to his neck. As a young man he saved the corks from the bottle of beer he drank for his supper every night and after eight years sold them and with this money started to make his fortune by successful stock jobbing.

Mr. Foscue was farmer general of Languedoc. He made much money out of the collection of taxes and suddenly was ordered by the government to raise a large sum of money at once. He pleaded poverty, and, fearing his house would be searched, dug a cave in the wine cellar and hid the money in it. Soon afterwards he himself was missed, sought for but not found. Later his house was sold and in altering it or tearing it down the workmen discovered the door leading to the cave. On breaking it open—it was closed by a spring lock—they found the dead body of Foscue lying among the vast riches he had accumulated.

Thomas Milbourne was not a true miser. He spent nothing on himself, though he gave much to others. He started life as a farmer's servant and saved two hundred pounds. He purchased a small farm in Cumberland, lived alone, and did all his work himself. He clothed himself in rags, never shaved, and the contents of his house were sold at his death for less than ten shillings, though his estate amounted to one thousand pounds. Many promissory notes were found and it was discovered that he had lent many of his neighbors money on the easiest terms and never pressed anyone for payment. He had, however, the miser's habit of hiding money. He died in 1800 between seventy and eighty years old.

Samuel Stretch was another example of a man who deprives himself of things in order to obtain, by money, a posthumous

fame or at least remembrance. He died at Madeley in 1804 but at what age I have not been able to discover. In early life he served as a private in the army and took part in some real fighting. His occupation in late life was to carry letters and parcels to the towns surrounding Madeley and to do any little commissions his neighbors might give him. For years he admitted no one to his hut and lived entirely alone. His clothing was in tatters and over his shoulders he carried a bag into which he put bones, bits of leather, paper, rags and indeed all kinds of trash that he found in his wanderings. His linen consisted of two old shirts and a pair of sheets. He amassed quite a large sum of money but the exact amount is not recorded. He left money to purchase an additional bell for the village church, and set aside sums to pay for having it rung at nine o'clock on summer nights and eight in winter, a bell for the free school, an addition to the salary of the organist, to enlarge and repair the almshouse, and for clothing and educating two poor children and to his relatives he left two shillings and six pence each.

Thomas Cooke was born near Windsor in 1726 and died in London 86 years later. He is of particular interest to medical men because he was a classical example of dispensary abuse. All the gentlemen interested in reform of hospitals for the good of doctors' pocketbooks ought to read his life. They could quote him in their papers and speeches. His father was a wandering fiddler who died when Thomas was a little child, leaving him to the care of a grandmother. As a youth he showed one good quality: he lived on bread, water and apples to save money to pay a village schoolmaster to instruct him in reading, writing and arithmetic. He was employed as a porter by a dry salter and did his work so faithfully that his employer helped him to get a place as exciseman. After his appointment, the government sent him to oversee a paper mill as tax master and he studied the business so well that he soon found the proprietors were cheating the revenue. He said nothing till the master of the mill died; then he interviewed the widow, told her of the fraud and that he alone knew of it but, that if the government learned of it all her property would be seized to make up the loss, whereas if she would marry him, he would not reveal the secret. She married him. He continued the business but not successfully and then went into the sugar trade. Though he

was a successful man in a worldly sense, leaving at his death more than 127,000 pounds in consols, his whole life was one of penury, petty saving, and petty trickery. He made it a habit in order to get meals for nothing, to fall in a pretended fit in front of a house at dinner time and on recovering he would naturally be invited to share the meal, which he always did after the proper amount of protestation. Often he would pretend to these kind people that they had saved his life, would make a great fuss over them and tell them he intended to remember them or their children in his will. Not a few of these people learned he was rich, did not object to being remembered in wills, and thought to increase their chances by making gifts to him. He received in this way geese, turkeys, roasting pigs, hares, pheasants, and sometimes a dozen of fine wines. One man, a poor relation, occasionally sent him small presents of butter. This angered Cooke who said to him "why send me such dribblets, you who are to get thousands and thousands at my death. Send me a firkin." The firkin and several more were sent, but neither this legacy nor any of the others came to reality. He used ink to black his shoes but never paid for it; instead he begged it, sometimes pilfered it, carrying a bottle with him for the purpose. He was very fond of cabbage and used some ground around his house to grow it on, getting the manure by going out in the road with a little shovel and basket and gathering up the horse droppings. His parsimony toward his wife, who had been well cared for by her first husband, caused her death. He had one healthy pleasure. He was fond of horses, but fed them largely at other people's expense. He was a good talker and an interesting man and as he rode along the road he made it his business to meet a farmer driving a load of hay. He would get in conversation with him and meanwhile his horse would make a good meal of hay. Like many men shrewd in petty ways, he was easily deceived in matters out of his own line. Thus once when his horse was sick he was too mean to pay a horse doctor and asked advice of a quack who told him he must take thirty onions, drill a hole through each, put them on a string, put the necklace around the horse's neck and let it stay there. The expense of thirty onions was too much: he bought fifteen and when after many days, they had served their purpose, he took them to the servant and ordered her to make an onion porridge for the day's dinner.

His cook was well trained but that time she rebelled and the porridge was not made. He had no feeling of mercifulness towards animals and once purposely and horribly maimed a horse he intended to buy in order to reduce its price, pretending it had been injured by its own viciousness. Numberless stories are told about him, all illustrating his skill in petty scheming to save or make small sums of money. Doctors especially he tricked and cheated, among others the celebrated Dr. Lettsom. Even while on his death bed he quarreled and wrangled about medical fees. He asked the physician in attendance to tell him how long he might hope to live. The reply was six days. He flew into a real or pretended rage and blackguarded the doctor for taking his money when that was all he could do.

One of the characteristics of misers is the lack of the social sense, the self-centeredness, the absence of the need of companionship. It is true that not a few of them have been married, but in all instances the wife seems to have been taken simply because she was cheaper than a servant or because her money was desired. It is rather remarkable, therefore, that there is alleged to have existed in London at one time a miser's club called the "split farthing club." As everyone knows, London is the club city of the world and clubs and societies of all kinds of impossible people for all kinds of impossible purposes flourish there, so that one need not be astonished at anything in the club line. I do not wish to be held responsible for the trustworthiness of my source of information concerning the matter but refer you to "A Compleat and Humorous Account of all the Remarkable Clubs and Societies in the Cities of London and Westminster" published in London in 1756. You must judge for yourselves whether the book was written in good faith or whether it is an example of labored and, in places, rather dirty wit. I have a great deal of doubt about there ever having been a "split farthing club" because it would be entirely out of consonance with the character of a miser to have much to do with anyone in any social way. They have almost the egoism of the insane who, barring the epileptics, never combine either for social intercourse or to conspire to injure.

Neither intellect nor education prevent the development of the miserly instinct. The tinker and the man of science alike fall victims. No veneering of acquired characteristics can prevent

the real man showing through. Even physicians may suffer from the disease, but I am glad to say that the number is small. Jacques Dubois, alias Jacobus Sylvius, immortalized in the Sylvian fissure is the most notorious example. I crib my biographic sketch from James Moores Ball's "Andreas Vesalius the Reformer of Anatomy." Sylvius was the most popular medical teacher in Paris when Vesalius was a student there—popular only as a teacher not as a man. He was born near Amiens in 1478. As a young man he was noted for his scholarship, especially in philology. He gave up the study of languages to make money at medicine. There seems to have been more money in physic in those days than now. He was the first man in France who taught from the cadaver, but he regarded Galen's writings as infallible and if the cadaver showed structures unlike Galen's description, the fault was not in the book but in the dead body or perchance, and this argument was not made by Sylvius alone, human anatomy had changed. Sylvius was a man of vast learning but he was in character contemptible, rough, coarse and brutal. He never used a fire to warm himself but took violent exercise in his room instead. Once in his life his friends found him hilarious. Asked the cause, he said he had just dismissed his "three beasts, his mule, his cat, his maid." He was notoriously rigid in making his students pay their fees. He was violent and vindictive in argument and jealous of the fame attained by others. In later years he was opposed by Vesalius and spoke of him, not as Vesalius but as Vesanus, a madman. He is said to have been the first man who used colored injections in anatomical investigation. He was thought to have amassed great wealth, but little was found after his death and what there was was hidden in secluded places. Years after his death when his house was torn down many gold pieces were found. He died in 1555 in the seventy-seventh year of his age and was buried, as he had wished, in the pauper's cemetery. His epitaph runs in English.

"Sylvius lies here, who never gave anything for nothing:
Being dead, he even grieves that you read these lines for nothing."

Let me refer to a man who endured the reputation of being a miser in order to save money to found and aid charitable institutions. He was Thomas Guy, founder of Guy's Hospital. He

was the son of a lighterman and coal dealer in London and was himself bred (I use the word the old books use) a bookseller. He began trade in London with 200 pounds. He speculated in South Sea stock in 1720 and made an immense fortune. He always dined alone, using a proof sheet or a newspaper for a tablecloth. Vulture Hopkins called on him once, saying he wanted to learn frugality from him. "We do not need a light to talk about that" said Guy and put the candle out. Hopkins left, saying he needed no further instruction. But Guy was not in any sense a miser, notwithstanding the mean life he led and the personal sacrifices he made. He saved for charity. While still living he gave 18,733 pounds to found Guy's Hospital and left it when he died 219,499 pounds. He gave much money to other charities and no hidden gold pieces or bank notes were found in rat holes, buried pots, or the like. He died in 1724, aged eighty-one years.

I could continue indefinitely relating stories, for literature is full of accounts of these people but it would be mere repetition. In all there is the same clinical picture, in all there is one and the same type of man or more rarely woman. Possibly in the future, if feminism becomes a reality and not a crazy dream, the rareness in women may cease. When we try to learn the true psychology of the miser, we find there is but little data: authors are so interested in the melodramatic and the eccentric, in the queer and the squalidly picturesque events of the lives that they pay attention to nothing else. I have not been able, *e. g.*, to find a detailed and accurate account of the boyhood of any miser and yet such knowledge is of vital importance in studying the psychology of any type of man, because it is probable that if we really knew a boy's mental makeup soon after puberty, if we could see into his mind at this time, when he breaks through the shell of childish, unconscious egotism which has kept him a being detached from the outside world, a thing separate and apart, unknowing that he has a personality, and realizes, as every thinking boy does (the others are not worth bothering about) that he is a part of and yet distinct from the universe, we could foretell his mental history for the rest of his life, because during this time his hitherto formless mind rapidly takes shape, becomes crystalized. The mental potentialities which determine character are all present in boy-

hood, but we are not alert enough to grasp their significance when they now and again come fleetingly to the surface and hence can not tell which will remain potential, which will become kinetic. Furthermore though all misers are more than superficially alike, all have the same predisposition and offer the same soil, all have the passion for acquiring useless money and almost all the habit of hiding it, it is probable that there are several exciting causes and what they are it is usually impossible to discover. Certainly a person who presents any characteristic all his life differs somewhat from the one in whom it appears only in maturity and yet this difference is just what we find in misers. Several of them as youths and young men showed no sign of abnormality and then suddenly underwent, in the absence of any known reason, a complete change of manners, habits, disposition, just as the insane do, while others showed miserliness even during adolescence. In some cases the explanation doubtless is merely that the development of character was slow, but in any disease of sudden onset we expect to find an external exciting cause. Mental ability does not protect against miserliness, for there have been many men of mental power far above the average who have suddenly lost interest in intellectual pursuits, without any other evidence of insanity, and have lived the lives of misers. So scanty is our data, therefore, that to draw conclusions about these people we must often, and sometimes doubtless erroneously, draw inferences from trifling incidents because more serious and important things are not recorded.

In classifying the type it must be remembered that a miser is not merely a man extremely stingy: miserliness is not mere avarice. Again a man may, as in some of the cases related, deprive himself of every thing except the barest necessities, even live in filth, and yet not be a miser, because his motive is purely altruistic, purely a desire to do good during life or after death. Nor have all such men had fame as their motive. Some of them have been careless and unconcerned as to whether or not they were remembered or forgotten and acted from motives of the purest generosity or from an imperative sense of duty. A genuine miser is a man who collects money for the mere enjoyment of its possession, not for what it buys either in pleasure or power, but just as a magpie collects things. Indeed a fanciful

psychologist might explain miserliness on the grounds of atavism and say that it was a reversion to ancestral type, a throw back to our alleged bird ancestors, just as some weakly imaginative criminologists explain crime as a reversion to the primitive man. The one is no more unjust to the magpie than the other is to the savage.

Several things strike one in studying these people, especially the great age to which many of them live. Indeed if anyone wanted an argument to prove that we of to-day have become too dainty and nice and ladylike in claiming that clean food and plenty of it, clean houses with much light and ventilation, frequent bathing, in short all the things the worshipers of the god hygiene demand, are necessary for the maintenance of health and even of life and that rather rougher life would strengthen the race he has at hand a plausible argument in the longevity of misers. Of course the real explanation is that only the sturdy can be misers and survive. Their longevity does prove, however, that given a man of strong constitution he can survive under the hardest conditions of life. It does prove that congenital ability to resist stress has more to do with long life, not with the mere saving for a few years of useless lives, than any external circumstance. Surely few men leading the most hygienic and sheltered lives, and the terms have unfortunately become in the minds of many people synonymous, live as long as many misers who expose themselves to so many of the evils that we are taught always kill.

Social standing has no bearing on the causation of miserliness, since its victims are found in all ranks of society. Children of rich and poor, learned and unlearned, of good and bad all alike may come to the same end and disbelievers in heredity may use this as an argument but they should remember that externals play little part in heredity. Need of money seems rarely if ever to be an exciting cause in starting from necessity what later becomes a fixed habit. That the condition is pathological goes without saying but it is not an insanity in the technical, restricted meaning of the word. Though it often leads to a blunting of the moral sense and even to cruelty it does not lead to intellectual stupidity nor to a delusional state of mind, though it narrows the circle of the intellectual life sometimes almost to a point, to one

idea. It is not an obsession because the obsessed are affected against their wills, fight the obsessions and are made unhappy by them, while misers enjoy. It is in a certain sense and to a degree a perversion of the esthetic sense, as is shown in the pleasure obtained in eating decayed food, and the enjoyment from shivering in a freezing room, and is in so far somewhat analogous to sexual perversion. That is to say, misers get pleasure from sensations which give the normal man pain. For example the man who washed his shirt in urine did not do so for economy's sake—water is too cheap—but I suspect he got a pathological pleasure from the urinous odor. This does not mean sexual pleasure, though the disciples of Freud would probably so interpret it. Miserliness differs, of course, from the true insanity in which there are delusions of poverty and the patient thinks he has not the wherewithal to get his bread.

I have tried to learn—because I believe that all constitutional disease stamps the external anatomy with its individual hall mark, only our eyes are not sharp enough often to see it—whether there is a characteristic physiognomy in misers, but without success. Most of those pictured in literature lived before the days of photography and the artists who drew the pictures to illustrate articles about them were more interested, I suspect, in creating striking illustrations and showing the melodramatic dirtiness of their clothes than in making physiognomically correct likenesses. Of course in those who were so celebrated as to be known to the people the pictures had to be more or less lifelike. The only conclusion that one can draw is that misers, like most abnormal people, do have casts of countenance which are unlike the ordinary types of faces. Many of them have the same look of furtive intensity that is common in the paranoiac, the religious maniac, and in many other dwellers in the borderland of insanity. They are almost all thin but often wiry. They are long faced and may have high or sloping foreheads. Their noses are frequently long and seldom broad at the opening of the nostrils.

In one minor mental characteristic they often resemble a certain type of chronic maniac in possessing a cutting, sarcastic and cruel sense of wit. Doubtless many of the witty sayings attributed to them are apocryphal, but many are true and even the false ones show that unconsciously the public have learned to associate a certain form of wit with miserly characteristics.

REMARKS ON THE CENTRAL REPRESENTATION OF SENSATION

BY WILLIAM G. SPILLER, M.D.

PROFESSOR OF NEUROPATHOLOGY IN THE UNIVERSITY OF PENNSYLVANIA,
FOREIGN CORRESPONDING MEMBER OF THE NEUROLOGICAL SOCIETY OF
PARIS, CORRESPONDING MEMBER OF THE SOCIETY OF GERMAN
NEUROLOGISTS AND OF THE SOCIETY FOR NEUROLOGY AND
PSYCHIATRY IN VIENNA

It is my object in this paper to present from personal observations extending over a considerable period certain facts relating to the representation of sensation in the spinal cord and brain. There will be discussed:

1. The location within the spinal cord of the fibers of pain and temperature and the importance of this location in surgical attempts to destroy the fibers of pain.
2. The separate course of pain and temperature fibers within the central nervous system certainly as high as the upper part of the pons and cerebral peduncles and presumably higher.
3. The presence of only pain and temperature fibers in the spinal root of the fifth nerve.
4. The separation of fibers of pain in the spinal root of the fifth nerve from fibers of temperature. This being true the assumption may be entertained that fibers of pain elsewhere in the central nervous system, though closely associated with fibers of temperature, do not occupy identical positions.
5. The possibility of fibers of heat having a separate situation from fibers of cold sensation.
6. The importance of the tractus spinothalamicus et spinotectalis as regards sensation, and the course of this tract so far as it is known. Evidence on this subject afforded by clinico-pathological investigation.
7. The representation of the three divisions of the trigeminal nerve in the spinal root of this nerve.
8. The representation of pressure sensation of the face in the facial nerve.

9. The position of sensory fibers within the inner capsule and the possibility that the different forms of sensation are separately represented in the parietal lobe.

I. THE LOCATION WITHIN THE SPINAL CORD OF THE FIBERS OF PAIN AND TEMPERATURE

It seems probable that the fibers of pain and temperature sensations within the spinal cord are contained within the tractus spinothalamicus et spinotectalis. A valuable case for determination of this question was one that occurred in my service at the Philadelphia General Hospital and was reported by me in 1905.¹ A small tubercle was found in the right lateral column at the extreme lower end of the thoracic cord, involving the area of Gowers' tract and the region median to it. About one half to one inch higher another small tubercle was found involving the left tract of Gowers and the adjacent region in which is contained the tractus spinothalamicus et spinotectalis. Sensation for touch was normal or nearly normal in the lower limbs, but sensation for pain was abolished or almost abolished in these parts. Sensation for temperature was almost lost in the lower limbs below the knees, and much impaired in the thighs and lower part of the trunk. The conditions were changed after a fall down a flight of stairs.

Another case² under my observation which showed that the fibers of pain and temperature must be represented in the anterolateral regions of the cord was one of thrombosis of the cervical anterior median spinal artery. Sensations of heat, cold and pain were greatly affected in the left lower limb and both sides of the trunk as high as the first rib on the left side and as high as the second rib on the right side. The anterior portion of the spinal cord in the cervical region was destroyed, and the fibers of Gowers' tract and of the tractus spinothalamicus et spinotectalis were implicated. The much greater escape of the right lower limb is to be explained by the posterior position of the fibers in the lateral column from the lower limbs. The degeneration was not equally intense in the anterolateral columns of the two sides. The lesion involved only the anterior part of the crossed pyramidal tracts and only slightly, and thus the tractus spinothalamicus et spinotectalis was implicated on each side in the lesion.

¹ Spiller, *JOURNAL OF NERVOUS AND MENTAL DISEASE*, 1905, p. 318.

² Spiller, *ibid.*, 1909, p. 601.

Somewhat contradictory to the view expressed above is the experimental work of Karplus and Kreidl.³ These investigators found that division of both halves of the cat's cord at different levels did not greatly impair pain sensation, and they concluded that the gray matter of the cord must play an important rôle in the conduction of sensation. We have, however, no evidence that in man long tracts exist in the spinal gray matter.

It thus seems probable that the fibers of pain and temperature occupy the tractus spinothalamicus et spinotectalis. In the spinal cord this tract is interior to Gowers' tract. In division of the anterolateral column of the cord for the relief of pain it is necessary that this tract should be divided, otherwise pain conduction may not be destroyed. The cut therefore must not be too superficial.

The literature fully justifies the assumption that the fibers of pain and temperature are situated in the antero-lateral columns.

2. THE SEPARATE COURSE OF PAIN AND TEMPERATURE FIBERS WITHIN THE CENTRAL NERVOUS SYSTEM

The fibers for the conduction of the various forms of sensation from the limbs, trunk and face are distinct from one another in the central nervous system probably as high as the optic thalamus, certainly as high as the upper part of the pons, as shown by an important case studied and first reported by me in 1909.⁴ The case was supposed to be one of multiple sclerosis. The symptoms which are of interest as regards sensation are as follows: The man had entire loss of sensation for heat, cold and pain on the entire right side of the body including the face as well as the limbs. He had slight impairment of sensation for heat but not for cold over the left forehead. Tactile sensation was entirely normal over the entire right side in the parts in which temperature and pain sensations were lost, and sense of position was promptly recognized in the right hand. Tactile sensation was entirely lost in the left side of the face but only in the distribution of the fifth nerve; pain and temperature sensations were normal in this region, excepting the slight impairment of heat sensation in the left forehead. The tactile loss did not extend over the scalp behind a line drawn vertically to the ear, or into the

³ Karplus and Kreidl, *Archiv für die ges. Physiologie*, Vol. 158.

⁴ Spiller, *Review of Neurology and Psychiatry*, February, 1910.

distribution of the cervical nerves on the left chin. Deep pressure was felt below the left eye, but it was not felt above the left eye. When the mouth was opened the jaw deviated markedly to the left. The left temporal and masseter muscles were completely paralyzed, and there was much flattening in the portion of the face normally occupied by these muscles, presumably from atrophy.

There was therefore the dissociation of sensation of the syringomyelic type (preservation of tactile sensation and of sense of position, loss of pain and temperature sensations) on the entire right side of the body, including the face and limbs, with an inverse type of dissociation of sensation to that of syringomyelia (preservation of pain and temperature sensations, except of heat sensation over the left forehead, with loss of tactile and pressure sensations, except of deep pressure sensation below the left eye, strictly confined to the distribution of the left fifth nerve, and with loss of function of the motor portion of the left fifth nerve.

It seems probable that the lesion in this case was in the left tegmentum of the pons. It implicated the central fibers of pain and temperature sensations coming from the entire right side of the body, probably those from the trunk and limbs being contained within the tractus spinothalamicus et spinotectalis within the left side of the pons, and those from the face possibly in the central tract for the face fibers nearer the raphe, as the central tract of the trigeminal nerve in the medulla oblongata is believed by some to be situated a little in front of the hypoglossus nucleus; but it seems improbable that the fibers of temperature and pain from the face should occupy a central tract distinct from that which contains the similar fibers from the trunk and limbs. Head and Holmes⁵ evidently believe that one central tract contains both sets of fibers. They say: "All impulses capable of generating pain become grouped together in the same path, and can be disturbed, simultaneously by an appropriate lesion of the spinal cord." What is true of the pain fibers from the body it seems to me probably is true also of the pain fibers from the face. Head and Holmes say that in the brain stem on their way to the optic thalamus "impulses underlying sensations of pain, heat and cold seem alone to run unaltered between the upper end of the spinal cord and the optic thalamus. They receive the ac-

⁵ Head and Holmes, *Brain*, Vol. 34, 1911.

cession of the regrouped secondary impulses from the face, which cross to join the specific paths for pain, for heat or for cold." Thus they imply that pain impulses from the face join in the same tract those from lower levels.

The lesion, in the case I am describing, involved the left sixth nerve and the left acoustic nerve or its central tracts passing to the right side of the brain stem. It involved completely the motor portion of the left trigeminal nerve in or near the motor nucleus; it involved also all the fibers of touch in the left trigeminal nerve, and slightly those of heat coming from the left forehead. The sensory root of the trigeminal nerve divides soon after entering the pons; some of its fibers pass a short distance upward to terminate in the sensory nucleus within the pons, whereas many of the fibers from this root pass downward into the medulla oblongata near the periphery of the pons. The ascending fibers probably are those of touch, and were implicated, together with the motor fibers of this nerve, in the lesion; whereas those which descend near the periphery of the pons probably are concerned with temperature and pain sensations, and these evidently were not implicated in the lesion. The central fibers from the nucleus accompanying the spinal root of the trigeminal nerve cross the raphe at different levels, and were not seriously implicated in the lesion. Whatever explanation may be offered for the peculiar sensory disturbance in the distribution of the left fifth nerve, it seems clearly demonstrated by this case that the tactile fibers separate soon after entering the pons from the pain and temperature fibers of this nerve.

The complete paralysis of the motor fibers of the left fifth nerve places the lesion, if intramedullary, clearly at the level of the entry of this nerve into the pons, as the motor portion has no descending root. That the lesion must have been intramedullary is shown by the other symptoms. The escape of sensation in the left side of the face, except for tactile irritation, shows that the whole fifth nerve was not implicated. The motor nucleus lies median to the sensory nucleus in the pons. The only possible conclusion is that the motor part of the fifth nerve was implicated with the central tract for tactile sensation median to the position of the sensory nucleus and the descending spinal root. This conclusion seems almost as positive as though a microscopical examination had been made. A lesion in the left tegmentum of

the pons would therefore explain the symptoms of the left side of the face mentioned, it would not have to extend far downward to implicate the left sixth nerve (for this nerve also was affected), and it would implicate the central tract for the fibers of pain and temperature supplying the right side of the body and face.

What I especially wish to emphasize is that this case demonstrates that the central fibers for touch in the face are distinct from those for pain and temperature in the face as high at least as the upper part of the pons, and it is presumable that the same condition exists higher than this level.

3. THE PRESENCE OF ONLY PAIN AND TEMPERATURE FIBERS IN THE SPINAL ROOT OF THE FIFTH NERVE AND (4 AND 5) THEIR SEPARATE REPRESENTATION

It is evident that the descending spinal root of the fifth nerve contains only fibers of pain and temperature, so much can be inferred from this case, but more conclusive as regards this point are the cases of occlusion of the posterior inferior cerebellar artery with microscopical examination. The descending spinal root is implicated in the lesion in these cases, pain and temperature sensations are often affected in the fifth nerve distribution on the side of the lesion, but tactile sensation in this distribution remains intact. Pain and temperature sensations are not invariably lost together from a lesion of this character. In one of the cases of occlusion of the posterior inferior cerebellar artery reported by me⁶ in 1908 pinprick sensation in the face was normal, and a part of the spinal root of the fifth nerve was not implicated in this lesion. Unfortunately temperature sensation was not tested in this case, as the man lived only a few days after entering the hospital, and this test was neglected at the first examination. It is possible that temperature sensation might have been found altered. I make this statement because of a case I have studied recently in which pain sensation in the distribution of the fifth nerve was lost when temperature sensation in the same distribution was intact. This case demonstrates that the fibers of pain sensation must be distinct in the spinal root of the fifth nerve from those of temperature. This conclusion is of great importance, as when a central lesion disturbs pain sensation

⁶ Spiller, *JOURNAL OF NERVOUS AND MENTAL DISEASE*, 1908.

anywhere it usually disturbs temperature sensation also in the same parts, and if the conclusion be justified that pain and temperature fibers in the spinal root of the fifth nerve occupy different areas, it is presumable that the fibers conveying these sensations from all other parts of the body are also separately situated in the central nervous system. In the case of disturbed sensation in the two sides of the face referred to at length above, slight impairment of heat sensation but not of cold was detected over the left forehead. In this distribution pain sensation was not altered. We often find that cold sensation may be recognized when heat sensation is lost from a lesion of the central nervous system, or vice versa, or both forms of temperature sensation may be recognized only as one, either heat or cold.

One patient of mine, for example, who had symptoms indicative of a lesion of the brain stem below the optic thalamus complained of his left lower limb being so cold that he "thought it would freeze," and his wife would wrap him in blankets. When he took a cold bath the cold water felt like boiling water to the left lower limb and the left side of the trunk. Warm water in these parts felt hot. When I applied a cloth wet with cold water from the spigot to the left foot he said it felt as though it were burning the foot and he jerked the foot away. Pinprick in the left foot produced a sensation that he could not distinguish from burning or tingling.

I am inclined to believe not only that fibers of temperature sensation are distinct from those of pain, but that there may even be fibers for the conduction of heat stimuli distinct from those for the conduction of cold stimuli. This may seem an unreliable refinement of dissociation, but we assume that there are fibers in the central nervous system elsewhere highly differentiated for special function. We assume, for illustration, that there are the so-called "pupillary fibers" in the optic nerve which convey impressions of light stimuli that cause contraction of the pupil and are not recognized as light sensation. We are taught also that the pupillary reaction is not always the same for stimulation by daylight as for stimulation by electric light. Could there be greater differentiation than this?

Head and Holmes support the idea of special fibers for heat sensation and special fibers for cold sensation within the spinal cord. They say: "In the same way, sensibility to heat or to cold

may be lost independently of one another, showing that all the impulses upon which they are based have been sorted out into two functional groups, each of which passes by a separate system in the spinal cord" (*l. c.*, p. 107). They further say that from a lesion in the brain stem below the optic thalamus pain, heat and cold sensibility may be affected together, but any one of them may escape or be affected alone. "Thus von Monakow described loss of sensation to heat and cold without analgesia, and in the case recorded by Mann and later by Kutner and Kramer sensibility was lost to pain and to heat, whilst that to cold was unaffected."

The case to which I have referred which demonstrates that the central fibers for pain in the face are distinct from those for temperature in the descending spinal root of the trigeminal nerve is as follows:

H. G. White, 44 years of age, is in my service (Oct. 1914) at the Philadelphia General Hospital. The disturbance of sensation in the face began about March 28, 1914. He had been in a barbershop and after he left the shop the left side of the face felt numb. He attributed this at first to soap which the barber might have left upon the face, but washing the face did not remove the sensation. The paresthesia of the left side of the face thus began suddenly, and has always been limited to the trigeminal distribution. Its onset was not accompanied by anything resembling an apoplectic stroke. The sensation has been one of stiffness, of burning, of numbness. It has never been felt in the lower part of the face in the distribution of the cervical nerves. He has not been able to stand a fly on the left side of the face, as it "seems to tickle all over," but he can stand a fly on the right side of his face. If a fly crawls over the left side of the face the tickling sensation extends over the left side of the face and into the throat. The sensation is like that of tickling in the sole of the foot. Tickling the left side of the face by light contact with the end of a cloth is very annoying to him. A fly was caught and made to crawl over the left side of the face, and he distorted his face and showed signs of great discomfort. When the fly crawled to the right side of the face he had no discomfort. If he suddenly hears the sound of a whistle he has a choking sensation in his throat. This over-reaction to sensory stimuli from a lesion probably in the medulla oblongata is very interesting and hardly in conformity with the statement made by Head and Holmes that the over-reaction to sensory stimuli can be attributed alone to disturbed activity of the optic thalamus.

The left eye felt as though it were drying up. He had at first

some pain over the left eye only in the distribution of the supra-orbital nerve. Soon after the numbness began he complained of choking sensation in the throat, his throat felt stiff and as though he had some obstruction in it, and this has caused him to cough. The left side of the face feels colder when he goes out of the house.

Analgesia for pinprick and deep pressure is complete (October, 1914) in the distribution of the left trifacial nerve and sharply confined to it. It extends in the scalp to the top of the head, then to the external auditory meatus, then to near the corner of the mouth and chin, and is limited by the midline of the face. The borders are fairly sharply defined. Tactile, heat and cold irritations are promptly recognized in this area and so acute is tactile sensation that he can tell at once when the pin penetrates the skin, but it causes no pain. Warm water is warmer in this area but cold water is equally cold on the two sides of the face. Immediately below the mouth on the left side pain sensation is diminished but not lost, as it is in the remainder of the trigeminal distribution. The mucous membrane of the left side of the mouth shows hypalgesia. The reflex from irritation by ammonia is normal in each nostril. The compass test shows impairment on the left side of the face. The motor branch of the left trigeminal nerve is not affected. The pain sensation of the tongue seems to be normal. The conjunctival reflex is lost on the left side and the corneal reflex is weak; these reflexes on the right side are prompt.

The left side of the soft palate moves much less than does the right side. The voice is hoarse, and the left vocal cord is paretic. He complains of a copper taste in the mouth, but a test shows no objective disturbance of taste.

The left pupil is a little larger than the right. The iridic reflex to light is impaired on each side, the contraction of the pupils in convergence is prompt. The tongue is protruded in the midline. Ataxia of the limbs is not present. The biceps, triceps, and patellar reflexes are about normal. The gait and station are normal. The pain and temperature sensations are normal in all the limbs. The Wassermann test was positive June 13, 1914.

The important features of this case are: The sudden onset of the analgesia in a syphilitic man, confined to the distribution of the left trigeminal nerve, without any other form of disturbance in this distribution; and the paresis of the left soft palate and left vocal cord. There is scarcely any doubt that the lesion is in the left side of the medulla oblongata implicating the descending spinal root of the left trigeminal nerve, and also the glossopharyngeus and vagus nerves. Especially important is the

evidence the case affords that such a lesion may disturb the fibers of pain in the trigeminal nerve without in the least affecting the fibers of temperature, except that warmth may feel a little warmer on the left side of the face. The lesion probably is vascular, hemorrhage or thrombosis, in the distribution of the posterior inferior cerebellar artery. So small a lesion as must be present in this case seems more probably hemorrhage.

The loss of deep pressure pain in this case is interesting. A pencil pressed deeply over the distribution of the left fifth nerve caused no discomfort. Head and Holmes found that a lesion in the brain stem below the optic thalamus "does not usually abolish completely all painful pressure. Thus it would seem that the grosser forms of pain and discomfort may possibly find their way by another path, if the usual one is closed."

Head and Holmes report two cases resembling this one of mine, except that in the first one the pain sensation was lost also in the half of the body opposite the lesion, and in the second the sensory disturbances of the body were not absent as in my case. In their Case 2 a lesion of the bulb caused analgesia of the opposite half of the body and same side of the face and yet the threshold-values of sensibility to heat and cold were equal on the two sides, although warmth seemed hotter and cold less cold over the analgesic parts. The onset in this case was as in my case in that there was no appearance of an apoplectic attack.

In their Case 3, one of occlusion of the posterior inferior cerebellar artery, pinprick sensation in the face on the side of the lesion was lost, but tactile sensation was normal. The threshold for the appreciation of heat and of cold was the same on the two sides, but the patient complained that all temperatures produced a duller sensation in the face on the side of the lesion. Pressure pain was the same on the two sides of the face. Temperature and pain sensations were affected in the body on the side opposite the lesion. Head and Holmes thus report two cases in which pain sensation in the face was lost but temperature sensations were preserved from bulbar lesions. It would seem as though pain fibers were more susceptible than temperature fibers. In their Case 1, one of injury high in the cervical region, pain sensation was lost in the right cheek and in the occipital region. The borders of the analgesic area were not sharply defined but merged gradually into parts of normal sensibility. No difference could

be discovered between the two halves of the face to measured tactile stimuli. Sensibility to heat and cold was not disturbed. The diagram accompanying the report of this case seems to indicate that the analgesia of the right cheek was confined to the distribution of the cervical supply, although this statement is not made by the authors. It seems to have been analgesia without impairment of touch and temperature sensations from a lesion of peripheral neurones. A similar condition has been obtained by Byrnes in some cases after injection of the Gasserian ganglion with alcohol, in that he has found that in recovery of sensation after such injection pain sensation may be the last to return (personal communication).

Head and Holmes do not give the tract for pain and temperature sensations. They say: "This does not necessarily mean that the impulses [for pain, heat and cold] are carried by uninterrupted fibers. It has been suggested by Long, Cajal, Kohnstamm that the nuclei of the *formatio reticularis* are intercalated in the course of the afferent conduction tracts. Such anatomical relay would not interfere with the above physiological conclusions, as it does not necessarily imply any regrouping of sensory impulses."

6. THE TRACTUS SPINOTHALAMICUS ET SPINOTECTALIS

We have some evidence as to the position of the fibers of pain and temperature; they probably lie in the tractus spinothalamicus et spinotectalis. This tract, according to Edinger and Petrén, is one of the most important secondary sensory tracts. Obersteiner⁷ says it is situated at the ventral part of the nucleus of the posterior colliculus of the corpora quadrigemina. Villiger⁸ makes it enter the median fillet in the pons, Marburg⁹ represents the tract in a series of drawings as occupying in the middle of the pons the lateral part of the median lemniscus and high in the pons as situated in the region of the lateral lemniscus, external to the superior cerebellar peduncle as the latter descends at the aqueduct of Sylvius.

The tractus spinothalamicus et spinotectalis is situated near

⁷ Obersteiner, *Anleitung beim Studium des Baues der nervösen Zentralorgane*, 5th edition, p. 474.

⁸ Villiger, *Brain and Spinal Cord*, translated by G. A. Piersol.

⁹ Marburg, *Mikroskopisch-topographischer Atlas des menschlichen Zentralnervensystems*.

Gowers' tract throughout its course as high as where the latter separates from it in the upper part of the pons to enter the cerebellum, and it is probably for this reason that disturbances of sensation have been attributed to lesions of Gowers' tract. In occlusion of the posterior inferior cerebellar artery the tractus spinothalamicus et spinotectalis is usually implicated in the lesion, and it is probably for this reason that the sensations of pain and temperature are affected.

According to Ziehen,¹⁰ who gives the best and most recent description, the fasciculus spinothalamicus et spinotectalis has been known only about a decennium. Its course in the spinal cord is known only in a general way. It is assumed that the tract arises in the cells of the posterior horn and possibly of the intermediate gray matter. The fibers probably cross in the white or gray commissure three or four segments above their origin, and pass into the lateral column. Some fibers probably do not decussate and these pass to the periphery of the lateral column of the same side.

The position of the tract in the lateral column can be given only approximately. It appears to lie immediately interior to Gowers' tract and is not sharply separated from it. Edinger was the first to distinguish the tractus spinothalamicus et spinotectalis and to give it this name. The fibers of this tract to the corpora quadrigemina and optic thalamus are united probably as far as the region of the corpora quadrigemina, and here some fibers of this tract possibly go to the corpus geniculatum mediale, others pass to the anterior and posterior colliculi of the corpora quadrigemina partly crossed and partly uncrossed, and the remainder pass to the optic thalamus. Some fibers of this tract occupy the anterior column of the spinal cord, but they are less important in man than in the lower animals.

The physiological significance of this tract is uncertain. The assumption that either this tract or Gowers' tract, says Ziehen, represents cutaneous sensation is not sufficiently based on anatomical, physiological or clinical evidence, and on account of the relatively small size of the former tract and on account of the course of the latter into the cerebellum such a relation to function becomes improbable. It is probable that the short connect-

¹⁰ Ziehen, *Anatomie des Centralnervensystems, zweite Abteilung, erster Teil*, p. 234. *Bardeleben's Handbuch der Anatomie des Menschen*.

ing fibers in the interior of the lateral column are concerned in an essential degree with sensation, but this must be determined by further investigation. Ziehen modifies, however, this statement in a very considerable degree. He explains the segmental disturbance of pain and temperature sensations by implication of a long tract, probably the tractus spinothalamicus et spinotectalis and ends by concluding that the question of the conduction of sensation in the spinal cord and medulla oblongata is almost as unsettled to-day as it was ten years ago.

There is no difference of opinion, says Ziehen, as to the ending of the majority of the fibers of Gowers' tract in the cerebellum, and the statements of Soelder and of Quensel as to a termination of some of the fibers in the corpora quadrigeminal region and optic thalamus probably result from a confusion of this tract with the tractus spinothalamicus et spinotectalis.

It is evident that the size of the latter tract has never been accurately determined, and it is not sharply defined from the surrounding fibers. It may be much larger than the statement of Ziehen would indicate.

One or two cases in the literature are important in regard to this subject.

The study by Economo¹¹ of a case in which a tubercle developed in the left side of the tegmentum of the pons convinced him that the fibers of temperature and pain in the pons must be lateral to the sixth nerve and in or near the lateral part of the median fillet, whereas the fibers of touch and deep sensation must be chiefly in the median part of the median fillet between the sixth nerve and the raphe. His location of fibers of temperature and pain in or near the lateral part of the median fillet conforms to the position of the tractus spinothalamicus et spinotectalis at this level.

In the case of occlusion of the left superior cerebellar artery reported by C. K. Mills¹² sensation in all its forms was fully retained on the entire left half of the body, but was lost to pain, extreme heat and extreme cold on the right half of the body; face, trunk, upper and lower limbs. Tactile discrimination was also greatly impaired, as shown by the compass test. Light touch was preserved. The senses of deep pressure and of position and

¹¹ Economo, *Jahrbücher für Psychiatrie und Neurologie*, Vol. xxxii.

¹² Mills, *JOURNAL OF NERVOUS AND MENTAL DISEASE*, February, 1912.

passive movement were normal on both sides and the man had normal stereognosis in each hand. Deafness in the right ear was complete. Occlusion of the left superior cerebellar artery was found. Among other lesions at the level of the mesencephalic root of the fifth nerve were degeneration of the left lateral lemniscus, of the superior cerebellar peduncle except of a



Photograph showing at *A* degeneration of the left lateral lemniscus, of the superior cerebellar peduncle except of a small portion near the median lemniscus, and of a small part of the tegmentum of the pons medial to the superior cerebellar peduncle. I am indebted to Dr. A. J. Smith for the photograph.

small portion near the median lemniscus, and of a small part of the tegmentum of the pons medial to the superior cerebellar peduncle. Here we must search therefore for the fibers conveying the sensations of pain, heat and cold, and this case shows therefore that these fibers are presumably in the tractus spinothalamicus et spinotectalis.

7. THE REPRESENTATION OF THE THREE DIVISIONS OF THE TRIGEMINAL NERVE IN THE SPINAL ROOT OF THIS NERVE

In 1901 Dr. C. H. Frazier and I¹³ reported the results of experimental work on the sensory root of the fifth nerve. He attempted to cut the sensory root near its entrance into the pons and to save the motor root, and in so doing some of the fibers of the sensory root next to the motor root escaped. The central nervous system from two of the dogs employed I found especially useful for microscopical study. The external portion of the sensory root at its entrance into the pons was much degenerated and the dorsal part of the descending spinal root was greatly degenerated. From this it may be concluded that the fibers of the external portion of the root, which probably represent the second and third divisions, are continued downward in the dorsal portion of the spinal root of the fifth nerve.

Bregman¹⁴ obtained results from experiments on the rabbit very similar to our results obtained in the dog. In his cases, where the descending spinal root of the fifth nerve was fully degenerated, the sensory root at its entrance into the pons was also completely degenerated; but where only the ventral portion of the spinal root was degenerated, only the medial portion of the sensory root at its entrance into the pons was degenerated, and where only the dorsal portion of the spinal root was degenerated, only the external portion of the sensory root at its entrance into the pons was degenerated.

An important paper on the representation of the three divisions of the fifth nerve in the spinal root of this nerve has been written within the past few years by van Valkenburg.

Eisenlohr many years ago concluded from a clinico-pathological case that the three divisions are not in the same areas in the spinal root, and that the third division terminates in the more proximal part of the root.

Wallenberg later in a case of complete anesthesia of the buccal mucous membrane found the proximal part of the spinal root, and especially its dorsal portion, the most degenerated.

Van Valkenburg states that Bregman's observation that in

¹³ Spiller and Frazier, University of Pennsylvania Medical Bulletin, December, 1901.

¹⁴ Bregman, *Obersteiner's Arbeiten*, Vol. i, 1892.

¹⁵ Van Valkenburg, *Monatsschrift für Psychiatrie und Neurologie*, Vol. xxix (1911).

rabbits the first division terminates more distally in the spinal root than the other two divisions, and that the third division terminates in the frontal part of the root and occupies the dorsal portion in a transverse section, was confirmed by Bochenek.

Van Valkenburg refers to the literature cited above in reporting a case in which there was anesthesia of the first division. The spinal root was degenerated in its most distal portion in the third cervical segment. At the level of the dorsal vagus nucleus a small area in the dorsal portion of the spinal root contained medullated fibers and gradually increased in size in more frontal sections approaching the sensory nucleus. From this case van Valkenburg concluded that the distal portion of the spinal root contain essentially fibers from the first division and these fibers occupy the ventral portion of the root in transverse sections in more proximal regions. The second and third divisions occupy the dorsal portion of the spinal root and terminate in the more proximal parts of this root and in the sensory nucleus in the pons. This view however has been disputed by some investigators.

I have examined two cases of syringomyelia with great disturbance of pain and temperature sensations but not of tactile sensation in the face. No other cranial nerve than the trigeminus was affected. It is presumable that a lesion in the medulla oblongata implicated the spinal root of the fifth nerve. The greatest impairment of sensation was in the distribution of the first division of the nerve, the forehead; the least impairment was in the distribution of the third division, the chin. In one of the two cases this arrangement was very striking. These cases would seem to show that the first division of the fifth nerve descends lower than the other two branches, and that the third division descends the least in the spinal root. Opposed to this view is that in which the representation of the spinal root in the face is in gradually diminishing circles as the anesthesia progresses.

The evidence of separate representation of the three divisions of the fifth nerve in the spinal root is of great value, and it seems not only that each division is represented in large measure separately, but that in this separate representation of each division in the descending spinal root the sensations of temperature depend on different fibers than do those of pain.

8. THE REPRESENTATION OF PRESSURE SENSATION OF THE FACE IN THE FACIAL NERVE

Head has shown that deep sensation of the body has a different peripheral nerve distribution from that of superficial sensation. It has seemed to me probable that the deep sensation of the face depends in part at least on the integrity of the seventh nerve. I¹⁶ reported briefly in 1906 two cases in which the pressure sensation was preserved after removal of the Gasserian ganglion, and I discussed this subject in a paper published in 1908 with a report of two cases. These two cases were referred to in a paper by Ivy and Johnson.¹⁷

In some cases of removal of the Gasserian ganglion for tic douloureux all sensations, including that of pressure, is lost, at least for a period, following the operation, but in the two cases of removal of the Gasserian ganglion for tumor which I observed, preservation of pressure sensation persisted with loss of other forms of sensation in the distribution of the fifth nerve. In some if not in all of these tumor cases in which the ganglion is implicated removal of the ganglion or of a large part of it does not seem to be followed by complete loss of pressure sensation in the distribution of the fifth nerve. This preservation of pressure sensation was very remarkable in my two cases, in one of which a large part of the ganglion had been removed, and was observed also in a case of Dercum, Keen and Spiller. In the case of Hofmeister and Meyer, after the operation light touch usually was not recognized, but pinprick was felt somewhat better without distinction of head and point; this may have been caused by preservation of pressure sensation.

In my first case before any operation tactile sensation was completely lost in the entire distribution of the right fifth nerve, provided little or no pressure was produced. Sensation of pain was lost in the same distribution. At the necropsy nothing could be seen of the Gasserian ganglion, as its position was occupied by a tumor. The right fifth nerve was enveloped in a tumor at its entrance into the pons, and the tumor covered the pons at the entrance of the fifth nerve into the pons, so that the fibers of this nerve became a part of the tumor immediately beneath the

¹⁶ Spiller, *JOURNAL OF NERVOUS AND MENTAL DISEASE*, 1906, p. 736, and *American Journal of the Medical Sciences*, November, 1908.

¹⁷ Ivy and Johnson, *University of Pennsylvania Medical Bulletin*, May, 1907.

pons. The nerve was completely degenerated at its entrance, as shown by the Weigert hematoxylin method.

In my second case tumor was found about the right Gasserian ganglion, and a portion of the tumor and ganglion were removed. After the operation touch was not felt in the distribution of the three branches of the fifth nerve unless it were made with slight pressure, when it was recognized. The right conjunctiva was anesthetic. At the necropsy a tumor was found occupying the position of the Gasserian ganglion and no distinct ganglion tissue could be found by the naked eye. The sensory root of the right nerve was intensely degenerated, as shown by the Weigert method, and nerve cells and nerve fibers of the ganglion found embedded in the tumor were much degenerated.

It is true that in some cases excision of the Gasserian ganglion causes complete loss of all forms of sensation, but this fact does not make the transmission of certain forms of sensation through the facial nerve impossible. It is possible that the deep sensation of the facial nerve may be temporarily interfered with by operation on the trigeminal nerve. Twisting out peripheral branches of the latter causes much swelling of the face, and probably thereby some impairment of the function of the seventh nerve. Excision of the Gasserian ganglion causes swelling of the axis cylinders and medullary sheaths of the peripheral branches of the trigeminal nerve, and if these are intimately associated with branches of the facial nerve this swelling may also interfere temporarily with the function of the latter. Destruction of the sensory root of the trigeminal nerve by a tumor affords better opportunity for testing the sensation of the face, than does destruction of some part of this nerve by operation, at least in the period immediately following operation. There is great need of further observation regarding the preservation of pressure sensation in relation to lesions of the trigeminal nerve before we can arrive at any positive conclusions.

In a case of mine of loss of tactile sensation in the distribution of the left fifth nerve, referred to in this paper, deep pressure sensation was preserved below the left eye where tactile sensation was lost. In a case reported by van Valkenburg, also referred to in this paper, superficial sensation was lost in the first division of the fifth nerve with degeneration of the descending spinal root, but rubbing and pressure were recognized in this area.

In their study of the pressure sensation of the face Maloney and Kennedy¹⁸ came to the conclusion that the fifth nerve must be regarded as the essential path for those impulses from the face which affect consciousness as sensations of pressure-touch; that after removal of the Gasserian ganglion pressure-pain may persist unimpaired in the face and tongue, but never in the eye; that the seventh nerve contains no pressure sense fibers distal to the Fallopiian canal, and that the sympathetic fibers subserve a general crude sensibility to pressure-pain.

9. POSITION OF SENSORY FIBERS WITHIN THE INNER CAPSULE

While we have satisfactory evidence that different forms of sensation are represented separately in the central nervous system as high as the basal ganglia, we have little to lead to a positive conclusion that the same is true of sensory representation at higher levels. It is presumable that the same rule holds good.

From two cases that I have studied, one with Dr. Dercum¹⁹ and one with Dr. Camp,²⁰ I believe that sensation, not including the special senses, is represented at the posterior part of the posterior limb of the internal capsule, as Charcot held, and that sensory fibers are not intimately intermingled with motor fibers in the posterior limb of the internal capsule. The separate localization of motion and sensation in the cerebral cortex is in support of this view, for it is impossible to understand why the sensory and motor fibers should be distinct as high as the subthalamic region, should mingle in the internal capsule, and separate again in the cerebral cortex. I can obtain no evidence however that the different forms of sensation are separately represented in the posterior part of the posterior limb of the internal capsule, but I should not be surprised if such evidence were obtainable later.

Wenderowic,²¹ from the recent study of a brain with the Marchi method, concludes that the sensory fibers do not occupy a separate region in the internal capsule but are spread over a great part of the capsule, and partly are mingled with the motor fibers, although the latter occupy a more clearly defined area and

¹⁸ Maloney and Kennedy, *Brain*, September, 1911, p. 1.

¹⁹ Dercum and Spiller, *American Journal of the Medical Sciences*, 1902.

²⁰ Spiller and Camp, *JOURNAL OF NERVOUS AND MENTAL DISEASE*, 1912.

²¹ Wenderowic, *Archiv für Psychiatrie*, Vol. 55, No. 2, p. 486.

not the entire area occupied by the sensory fibers. Sensory fibers are found in both central convolutions in nearly equal number, but the motor area is limited to the anterior central convolution. The motor fibers thus both in the cortex and in the internal capsule lie within the sensory fibers.

There can now be little doubt that the parietal lobe is the great cortical sensory area; it is possible that sensation is represented in its different qualities in different parts of this lobe, and it is probable that different segments of the body are separately represented as regards sensation in the parietal lobe, the sensory area being near the corresponding motor area, as shown especially by the experimental work of van Valkenburg.

THE FOUNDERS AND WORK OF THE PHILADELPHIA NEUROLOGICAL SOCIETY

BY T. H. WEISENBURG, M.D.

OF PHILADELPHIA, PA.

PROFESSOR OF NEUROPATHOLOGY AND CLINICAL NEUROLOGY, MEDICO-CHIRURGICAL
COLLEGE; NEUROLOGIST TO THE PHILADELPHIA GENERAL HOSPITAL

It is fitting on an occasion like this which celebrates the thirtieth anniversary of our society, that a record should be made of those who thirty years ago came together to start an organization which has continued as one of the leading factors in the development of neurology in this country. The initial meeting to take into consideration the organization of a neurological society was held in the office of Dr. Charles K. Mills in November, 1883, and was brought together through a call issued by him to a few of those interested in neurology in Philadelphia. Those present at this meeting were Drs. Sinkler, Eskridge, Dercum and Mills.

On December 15, 1883, a meeting was held in the hall of the College of Physicians in response to a call to take into formal consideration the establishment of a Philadelphia Neurological Society. It may be of historical interest to give here the names of the nineteen who were present at this meeting: Drs. Granville L. Angney, Alice Avery, Alice Bennett, Lewis Brinton, A. P. Brubacker, Robert H. Chase, Francis X. Dercum, J. T. Eskridge, Robert A. Given, S. Preston Jones, I. N. Kerlin, James Hendrie Lloyd, Charles K. Mills, George Moorehouse, N. A. Randolph, Edward F. Reichart, D. D. Richardson and Wharton Sinkler. Of the nineteen at least nine (Drs. Sinkler, Eskridge, Moorehouse, Kerlin, Richardson, Jones, Randolph, Given and Angney) have died.

A word or two might be said of some of these. Dr. George Moorehouse is chiefly known to neurologists by his association with Dr. S. Weir Mitchell and Dr. W. W. Keen in their work

on diseases and injuries of the nerves done chiefly at Turner's Lane Hospital, Philadelphia, during the Civil War. Dr. S. Preston Jones who was for many years the first assistant physician at the Pennsylvania Hospital for the Insane, was a well-known alienist and medical jurist. Dr. I. N. Kerlin was the able and energetic superintendent of the Pennsylvania Training School for feeble-minded children at Elwyn, Pa., which institution was brought by him to a high state of efficiency. He was a frequent contributor to psychiatry, especially in its relations to the mentally defective.

Dr. Wharton Sinkler left behind him a name which is dear to all members of this society and one which is respected wherever American neurology is known. He later became the president of the society and of the American Neurological Association. He always took an active interest in our proceedings, as is evidenced by even a casual examination of the minutes of the society. For many years he was one of the most active members of the staff of the Orthopedic Hospital and Infirmary for Nervous Diseases. He was also one of the founders of the Epileptic Hospital and Colony Farm at Oakburn, Chester County, Pa., the development of which owes more to him than to anyone else. In 1903 he was president of the National Association for the Study of Epilepsy and the Care and Treatment of Epileptics. He was the author of important contributions on the subjects of infantile paralysis (anterior poliomyelitis), chorea, epilepsy, and neurasthenia and wrote one of the earliest papers on syringomyelia recorded in this country. The phenomenon usually spoken of as the great toe reflex was first described by him in the *Medical News* for December 1, 1888.

Although he was personally known to only a few of the older members, it is well worth while to recall Dr. J. T. Eskridge, who was the first temporary secretary of the society. Dr. Eskridge was a man of great ambition and powers of work. In a few years he made for himself a reputation as a neurologist in Philadelphia, contributing numerous papers to the neurological, pathological and other societies. Owing to his poor health he went to Colorado, where he remained until the time of his death. Although hampered by ill health his courage never left him and he soon made himself the most prominent man in neurology and

ophthalmology in the Rocky Mountain west, becoming well known as a medical teacher, writer, neurological consultant and medico-legal expert.

The first formal meeting of the Society was held January 28, 1884, in the Hall of the College of Physicians, 13th and Locust Streets, at which time a plan of organization was presented by a committee and adopted by the society. At this meeting Dr. I. N. Kerlin acted as temporary chairman and Dr. Eskridge as temporary secretary.

The first officers of the association in accordance with the recommendations of the organizing committee were Dr. S. Weir Mitchell, president, Dr. Mills and Dr. Kerlin vice-presidents, Dr. Lloyd, secretary, Dr. Brinton, recorder, and Drs. Eskridge, Sinkler, Brubacker and Jones, councillors. The first meeting of the society for scientific work was held February 25th, 1884.

Dr. S. Weir Mitchell accepted the first presidency on the urgent invitation of all those interested in the formation of the society and continued as president for six years. He was usually present at the meetings and contributed valuable material to the society's proceedings. He was looked upon by every member as a guide, councillor and friend and his personal influence did much to promote the society's welfare. Since the death of Dr. Mitchell, January 4, 1914, many memorials of him have been held, his character has been ably delineated by those who knew him best, and the story of his work in science, medicine and general literature has been fully told. The readers are referred to these biographical sketches and analyses for details of the life and character of this great Philadelphian.

From time to time in the proceedings of the society Dr. Mitchell directed attention to those neurological matters which had for him especial interest and which he made particularly his own by numerous contributions to medical literature,—injuries and diseases of the peripheral nerves and physiological conclusions drawn from their study, neurophysiological investigations of various kinds especially those relating to the cerebellum, and the therapeutics of the nervous system. Not infrequently in the society he urged his younger colleagues in their zest for physiological and pathological investigation not to lose sight of the real work of the doctor, which was to relieve suffering and cure

disease. The attention of the society was often directed by him to new clinical types and subjects presenting such features of dramatic interest as allowed both his literary and scientific tendencies full play.¹

Of those who were present at the organization of the Philadelphia Neurological Society three are with us this evening and have places on the scientific program of this celebration. The society has also seen fit to elect them as its chief officers for this anniversary year. They are Drs. Mills, Dercum and Lloyd. From the time of the inception of the society to the present they have been active spirits in promoting its welfare as they have that of American neurology in general. The evidence of this is ample in the society's minutes and in current medical literature.

It is not always best to leave the recounting of the work and merits of those who deserve such recognition until they have passed from among us. It will assuredly not be out of place on the thirtieth anniversary of our society to recall to its members and its friends the neurological work of those of its number who have continued with it during its entire existence and have contributed largely to the advance of neurology.

A study of the bibliography of Dr. Mills shows that in the period between 1875 and 1914 inclusive he made 301 contributions to the literature of the period, all of these with the exception of about a score neurological. Among these were three text-books or treatises, parts of many "systems" concerned with

¹ Among the biographical memoranda relating to Dr. Mitchell are the following:—

At a special meeting of the College of Physicians of Philadelphia, held on the day of Dr. Mitchell's funeral, January 6, 1914, a minute was passed and addresses were made by the president of the college, Dr. James C. Wilson, by Dr. James Tyson, Dr. W. W. Keen and Dr. George E. de Schweinitz. On March 31, 1914, a joint memorial meeting was held at the college by the College of Physicians, the American Philosophical Society, the University of Pennsylvania, the Library Company of Philadelphia, the Jefferson Medical College and the Academy of Natural Sciences, at which addresses were made by the president of the college, by Talcott Williams, Esq., of Columbia University, by Dr. William H. Welch, of Johns Hopkins University, and by Mr. Owen Wister. The above addresses have all appeared in a memorial volume which can be consulted at the principal libraries of Philadelphia and the country at large. Other biographical sketches of Dr. Mitchell have been published, among these one by Dr. Charles K. Mills in the *JOURNAL OF NERVOUS AND MENTAL DISEASE*, Vol. 41, No. 2, 1914; another by Dr. James J. Putnam in the *Boston Medical and Surgical Journal*, Vol. clxx, No. 22, 1914, and others by Dr. Edward Jackson in *Colorado Medicine*, November, 1914, by Dr. Beverley R. Tucker, published by Richard G. Badger, Boston, 1914.

nervous diseases, general medicine, therapeutics, diseases of children and medical jurisprudence and numerous monographs and special articles. A study of his writings shows that they would fall more or less into subjects included under at least five groups, —(1) cerebral, spinal and peripheral localization, (2) cerebral schema and mechanisms, (3) new symptoms, syndromes or types of nervous disease, (4) miscellaneous contributions, clinical, clinico-pathological and medico-legal, and (5) medical biography and history.

In no field of neurology has the work of Dr. Mills been carried further than in that of localization, especially cerebral localization, although a consultation of his writings shows that he has given much attention to the subject in its spinal, plexic and peripheral aspects. So numerous have been the contributions of our president to the subject of cerebral localization that reference can be made in the time at my disposal to only a few.

As early as 1879 he began to record instances of focal disease of the brain with localization phenomena, reporting then a case of tumor with comments indicating the psychic functions of the frontal lobe and callosum.

Dr. Mills early indicated the importance of cerebral fissures, at least certain of the main fissures of the cerebrum, as probable boundaries of functional areas. He recognized more than twenty-five years ago the fact that the motor region was largely in advance of the central fissure, a position now almost universally held especially since the work of Grünbaum and Sherrington on the gorilla. In conjunction with surgeons like Dr. Keen, Dr. Frazier, Dr. Hearn and others he has added to our knowledge of cortical localization as the result of faradic applications to the human cerebrum, such applications having been made with the view of assisting in the more exact circumscription of fields of operation. He demonstrated, among other things, on the human subject the centers for movements of the head and of the head and eyes, for elements in the movements of Darwin's muscles of grief, the widespread diffusion of the representation of movements of the muscles of the jaw and the differences in stimulation required to produce localized movements by electrical excitation, caudad and cephalad of the central fissure. With the writer and Dr. Stewart Rodman, he was I believe the first to

indicate the representation of synergic movements in the cerebellum by stimulation of the human cerebellar cortex.

Dr. Mills postulated the existence of an orientation center (higher cerebral vestibular center) in the temporal lobe and to him is largely due the development of the symptomatology of the parietal lobe, a fact referred to by Bruns of Hanover at the meeting of the seventeenth international congress in London (1913). His work on the physiology of taste and uncinate symptomatology is well known and received recognition from Dr. J. Hughlings Jackson, the pioneer in this field.

The record of one of his cases,—that on the localization of the center for word-hearing through the study of a case with lesions of the superior temporal regions of both hemispheres,—has become classical in the history of the subject of aphasia, but it is not necessary for me to tell the members of this society of his numerous additions to our knowledge of such subjects as aphasia and the cerebral zone of speech, tumors of the brain and of the spinal cord, and cerebellar symptomatology and localization.

Among the most important contributions to cerebral mechanisms and cerebral physiological topography are those which are contained in his expositions of various new schema of the zones and centers of the human cerebrum, and his discussions of the separate localization of the motor and sensory functions in the cerebral cortex, of the subdivisions of the areas of representation of cutaneous and muscular sensibility and of stereognosis, of the subdivisions of the posterior association area of Flechsig which he has designated as the concrete memory field, of emotional expression and its cerebral representation and of the cerebral tonectic apparatus and some of the clinical problems of cerebral tone.

Dr. Mills has contributed his share to the list of new symptoms, symptom complexes and clinical types of nervous disease. To Wilbrand the credit for first calling attention to macular hemianopsia is usually given, but as Dr. W. C. Posey points out, this is due to Dr. Mills (*The Ophthalmic Record*, May, 1908).

What is known as Korsakoff's disease, the combination of alcoholic polyneuritis with special psychic phenomena indicating cerebral involvement, was recognized by Dr. Mills in 1886, some time before the publication of Korsakoff's first paper.

To him we owe the clinical type known as unilateral ascending paralysis due to degeneration of the pyramidal tract. On this subject he published not only his original case, but other papers dealing with different types of both unilateral ascending and unilateral descending paralysis. Still fresh in the memories of our members is his report of the first case of occlusion of the superior cerebellar artery, presented both to this society and to the American Neurological Association (1912). In not a few instances he was among the first to call attention in this country to types of organic nervous disease which had been observed abroad and in some of these cases without knowledge of their previous record. This was true with regard to disseminated sclerosis, he having published a paper embodying the report of a case with necropsy and serial microscopic examination as early as 1879.

A fact brought out in the bibliography of Dr. Mills and by a knowledge obtained from a study of his writings is the wide range of practical subjects which his contributions cover—neurological surgery or surgical neurology, cerebral morphology, vasomotor and trophic affections, hydrophobia, tuberculous meningitis, multiple neuritis, poliomyelitis, myotonia and athetoid spasm, surface thermometry, electro-therapeutics, problems in electrical potential, hypnotism, hysteria, neurasthenia, psycho-therapeutics, mental overwork, massage and Swedish movements, systematized exercises as a therapeutic measure, occupation neuroses, the localization of brain tumors by roentgen-ray exploration, aphasia and its treatment by training, disorders of pantomime, the symptomatology of lenticular lesions, intradural root anastomosis, insanity in children and adults, criminal lunacy and the medico-legal aspects of nervous diseases and of insanity.

The contributions of Dr. Mills to medical biography and history have not been without their interest. Among biographical sketches written by him are one on Benjamin Rush and American psychiatry, one on Isaac Ray, the great alienist and medical jurist, and his recent tribute to his friend of many years, Dr. S. Weir Mitchell. His historical papers include the history of medical jurisprudence in Philadelphia, the history of neurology in Philadelphia from 1874 to 1904, and historical sketches of the Philadelphia Hospital for the Insane and the Philadelphia Alms-

house and Hospital from 1854. One of his favorite recreations is the study of the local history of one of the most interesting sections of Philadelphia which has for its center the place of his birth, the Falls of Schuylkill. The associations of this old village with the history of the University of Pennsylvania and the military history of the neighborhood from colonial times to the end of the Civil War are among his additions to local history. He is a member both of the Pennsylvania Historical Society and of the City History Society of Philadelphia.

Dr. Mills began his teaching at the University of Pennsylvania as early as 1877, his first connection with the University as chief of the clinic for nervous diseases dating back about three years before this time. Subsequently he filled various positions, psychiatric or neurological, until on the retirement of Dr. Wood he was elected to his present position in 1901. In 1892 he was made Professor of Mental Diseases and of Medical Jurisprudence, the latter under what was known as the "George B. Wood Foundation." While he held his professorship of jurisprudence he was for several years dean of the auxiliary faculty of medicine of the University of Pennsylvania. It is interesting in connection with the chairs of mental disease and medical jurisprudence to note in the first place that with the exception of a few cases demonstrated at the Philadelphia Hospital by Dr. D. D. Richardson for Dr. Isaac Ray to illustrate his lectures on mental diseases at the Jefferson Medical School, Dr. Mills' clinical lectures on insanity were the first of the kind ever given in this city and probably in this country and in his chair of mental diseases he gave the first formally organized instruction in psychiatry in the university after the time of Benjamin Rush. He inaugurated the clinical teaching of the medical jurisprudence of insanity and nervous diseases by illustrating his university lectures by the demonstration of cases of medico-legal interest at the Philadelphia Hospital.

It is not as well known as it should be in how many instances Dr. Mills has been the initiator or founder of medical movements of importance both neurological and general. Reference has already been made to the part played by him with others in the foundation and development of this society. His greatest work, not only for neurology, but also for humanity, according not only

to my own estimation, but to that of many others, was the founding of the nervous wards of the Philadelphia General Hospital in 1877, to which he has given unflagging attention and interest from their organization to the present time. His last work to broaden the field of American neurology was the initiation of the movement which has just been consummated to establish a Philadelphia post-graduate school of neurology largely on the basis of the opportunities afforded by these wards which have grown to such proportions that they are now recognized as one of the greatest neurological services in the world.

He was one of the founders of the Philadelphia Polyclinic. He took a prominent part in the movement which resulted in the organization of the Congress of American Physicians and Surgeons in 1888. At its first meeting in Washington in September, 1888, he was one of the four to whom the main addresses at the general meeting were assigned. His address, the subject of which was "Cerebral Localization in its Practical Relations," was published in its entirety, not only in the proceedings of the Congress but also in *Brain*.

Dr. Francis X. Dercum, who with Dr. Mills and Dr. Lloyd had the honor of being one of the founders of this society, from the beginning of his professional career was interested in neurology. He has written altogether 132 papers, 8 parts of text-books or systems, one text-book on nervous diseases and one on insanity. At first he was on the neurological staff of the University of Pennsylvania, becoming chief of the out-patient department of the University Hospital. He was called to fill the chair of nervous and mental diseases in the Jefferson Medical College in 1892, this position being created for him. The manner in which he has conducted this department has made it one of the most important and distinguished of this school. For twenty-five years he was one of the visiting neurologists to the Philadelphia General Hospital, to the reputation of which he has largely contributed. Much of his valuable work has been based on his neurological service in this hospital. He has been connected with many institutions in Philadelphia and its vicinity, having been at various times on the staff of the Pennsylvania Institution for Feeble-Minded Children at Elwyn, the State Hospital for the Insane at Norristown, Pa., the Orthopedic Hospital and Infirmary for Nervous Diseases of Philadelphia, St. Agnes Hos-

pital, the Jewish Hospital, the Hospital for the Chronic Insane at Wernersville, etc. He was the third president of this society and has been president of the American Neurological Association and the Philadelphia Psychiatric Society. He is the present chairman of the Section of Nervous and Mental Diseases of the American Medical Association. He is one of the few American corresponding members of the Neurological Society of Paris and the Neurological and Psychiatric Societies of Vienna and a member of the Royal Medical Society of Budapest.

An analysis of Dr. Dercum's bibliography shows that his papers divide themselves into certain groups. Early in his career he became much interested in anatomical and morphological research. His first paper in 1878 on the "Sensory Organs, Suggestions with a View to Generalization," embraced localizations in regard to morphology which were later formulated by Haeckel.

In a paper on the "Morphology of the Semi-circular Canals" (1879), Dr. Dercum pointed out that these structures were in all probability evolved from the lateral line system of a remote aquatic ancestor by the involution of dermal structures, a view which was subsequently adopted by other biologists. In an investigation on the lateral line system of fishes, he pointed out that the nerve hills in this apparatus had a structure identical with that of the macula acustica and that the lateral line system was a sensory apparatus like that of the semi-circular canals and was adapted to receiving coarse wave impressions, the waves in this instance having a lower rate than that of sound vibrations.

Later came papers by Dr. Dercum on the "Morphology of the Brain in Epileptics" and "Studies in Criminal and Racial Morphology."

Especially interesting were some of his findings as regards the transition convolutions between the occipital and parietal lobes in some cases of arrested development and in the brain of a murderer. Some of the anatomical conditions were as he pointed out comparable to those met with in the higher apes.

In 1884 Dr. Dercum described in connection with Dr. A. J. Parker² convulsive seizures which could be induced by artificial means, demonstrations of convulsions so induced being subsequently made at a meeting of the Philadelphia Neurological Society at the request of the president, Dr. S. Weir Mitchell.

² The contributions of Dr. Parker to cerebral morphology have been described at length in the presidential address this evening.

A full account of the character of these seizures and of the method of their production was published in the *JOURNAL OF NERVOUS AND MENTAL DISEASE* in 1884. About this time (1884) Edward Muybridge was conducting his epochmaking investigations by his method of successive instantaneous photographs of animals in motion. Mr. Muybridge seized the opportunity of making photographs of a subject in these artificially induced convulsions. These pictures have since become historic, this being the first time in which photographs were made of a person in convulsions.

At about the same time Dr. Dercum took part with Mr. Muybridge in the extensive investigation conducted at the University of Pennsylvania of animal locomotion by means of instantaneous photography. These included the study of normal and pathological human gaits and various manifestations of disease. The results were published by the University in a book on "Animal Locomotion," in 1888. This volume included a chapter by Dr. Harrison Allen on the locomotion of animals as illustrated by instantaneous photography and another by Dr. Dercum on locomotion in man, normal and pathological. The Muybridge studies are interesting not only because of the results achieved at the time, but because they were the forerunners of the modern moving pictures.

Dr. Dercum has always had a marked interest in the functional side of neurology. This is evidenced by the large number of papers he has published on functional disorders, especially on hysteria and neurasthenia, and in the last few years in psychoanalysis; although it is not to be understood that Dr. Dercum by any means favors Freudian psychology, he being, in fact, one of its most determined opponents. In 1903 he published a treatise on "Rest, Mental Therapeutics and Suggestion," this being one of the first to contain in the English language a comprehensive consideration of psychotherapy.

Dr. Dercum has written on practically every phase of mental disease. It is interesting to note in connection with the subject of his address this evening on "Nervous and Mental Diseases and the Newer Pathology," that as early as 1885 he advocated a theory of the toxic origin of mental disease. In 1901 he advanced a clinical classification of insanity which has since in large measure been adopted. In 1902 he published a paper on "Hypochondria"

in which he delimited this neurosis and pointed out its differentiation from the other great neuroses,—neurasthenia and hysteria. Dementia præcox has received his attention in various contributions of unusual value. During the present year (1914) he has given the profession an excellent text-book on mental disease which embodies the results of his experience in practice, in teaching, and in the study of current medical literature.

Allied with his interest in mental diseases has been his work on different forms of dystrophy, especially in regard to the ductless glands. One of his earliest papers concerned itself with myxedema. Few if any have contributed more of value to our knowledge of scleroderma, acromegaly, cretinism, primary neurotic atrophy and the different types of obesity.

In 1888 he described under the title of "A Subcutaneous Connective Tissue Dystrophy" a new symptom group to which he subsequently in 1892 gave the name of *Adiposis Dolorosa*, publishing the first case with necropsy in 1900. Many articles on the same subject in which full credit has been given to the original observation have appeared during the last twenty years.

To clinical neurology, to cerebral and spinal localization, and to the surgery of the nervous system Dr. Dercum's contributions have been numerous and important.

In 1889 he reported with Dr. J. William White two cases of spinal surgery, in one of which complete recovery from paraplegia ensued after operation for a localized serous meningitis, this being one of the earliest of the successfully reported cases and in 1894 he reported with Dr. W. W. Keen two cases of in-gravescent cerebral hemorrhage treated by the rare operation for this affection,—ligation of the common carotid artery.

The early development of optic neuritis in cerebellar lesions; lead poisoning with special chemical investigation; Wernicke's pupillary reaction in a case of pulvinar disease; colloid implication of the blood vessels of the spinal cord; metastasis of the thyroid gland to the spinal column—these are a few of the numerous neurological papers by Dr. Dercum worthy of special mention.

As early as 1886 Dr. Dercum wrote a paper entitled "Facts and Deductions Bearing Upon the Action of the Nervous System," this containing many views recently advocated by Crile of Cleveland. In 1896 he advanced a theory of neuronie action,

according to which the terminations of the collaterals, dendrites and neuroaxons of nerve cells had the property under stimuli of retracting or extending, thus separating or uniting with other nerve cells.

In recent years, Dr. Dercum in his writings on aphasia has ably espoused the views of Marie, in this connection reporting many cases with necropsy.

As a medical jurist Dr. Dercum's reputation is more than local, his contributions to legal medicine being widely known and quoted.

James Hendrie Lloyd, from 1881 up to the present has written 100 papers, 12 special articles or treatises in different systems or text-books, and one book on the medical jurisprudence of insanity.

Dr. Lloyd has by no means confined his energies to neurology, he being one of the most scholarly physicians in Philadelphia. For several years he was the able editor of the Philadelphia Medical Journal, now incorporated with the New York Medical Journal.

Dr. Lloyd has been on the neurological staff of the Philadelphia General Hospital, with the exception of two brief intervals, since 1887. He has long been a member of the medical board of the Training School for the Feeble-Minded at Elwyn, Pa., and of the Hospital for the Chronic Insane at Wernersville, Pa. He founded the neurological service at the Methodist Episcopal Hospital of Philadelphia. He was the fifth president of the Philadelphia Neurological Society and was president of the American Neurological Association in 1889.

In cerebral localization the work of Dr. Lloyd has been conspicuous. In 1888 he published a paper with Dr. J. B. Deaver on trephining in focal epilepsy, one of the earliest articles based upon physiological cortical experimentation. The cortex was exposed and certain motor centers located by faradic stimulation. In 1888 another paper with Dr. Deaver was based upon the application of the principles of localization to spinal surgery.

His bibliography shows that Dr. Lloyd has contributed largely to the symptomatology of brain and spinal cord tumors. In 1885 he published with Dr. Mills monographic articles on tumors of the brain and of the spinal cord in Pepper's System of Medicine, this being one of the earliest contributions on the modern aspects of this subject.

Among other notable neurological contributions of Dr. Lloyd are those on diseases of occupation and diseases of cerebrospinal and sympathetic nerves, published in the *Twentieth Century Practice of Medicine* in 1895 and 1897 respectively; porencephalon; cerebral decompression; the X-ray localization of brain tumors; the surgery of the brain in pituitary disease and the changes in the spinal cord due to pernicious anemia.

In the discussion of the subject of aphasia Dr. Lloyd has taken the ground that both sensory and motor defects are found in so-called motor aphasia and in general he has antagonized the views of the extreme localizationists.

A case of syringomyelia with complete microscopic study was early recorded by Dr. Lloyd, and among his most valuable contributions are those on the traumatic affections of the cervical region of the spinal cord simulating this disease. With Dr. Ludlum, in a paper on primary lateral sclerosis, he advocated the view that this disease is a clinical entity and that it is frequently due to syphilis.

The medical jurisprudence of both insanity and of nervous diseases has been enriched by the pen of Dr. Lloyd. His treatise on "Insanity; Its Various Forms and Its Medico-Legal Aspects" in Wharton and Stille's *Medical jurisprudence*, Vol. 31, 5th edition, is the most valuable work on the subject since the treatise of Ray.

Among the many noteworthy papers Dr. Lloyd has published on mental diseases is that on the metaphysical conception of insanity as shown in the terminology of psychiatry (1904). Others by him are those on faith cures (1886), hysteria (1883), moral insanity (1886) and the *Œdipus complex* in Hamlet (1911). Psychoanalysis as advocated by him is not of the Freudian school.

It would be unjust in reviewing the work of Dr. Lloyd not to refer to the unusual value of his contributions from the standpoint of scholarship and literary merit as well as from their scientific aspects. The charm of his style and his enthusiasm in presentation have never failed to awaken and fix the attention of the society.

The Philadelphia Neurological Society was the second of local neurological societies in this country. The New York Neurological Society was founded in 1872. In Boston in 1880 a society was founded which was at first known as the Boston

Medico-Psychological Society. This was not in name or scope a strictly neurological organization. It became known later as the Society of Psychiatry and Neurology under which designation it still flourishes. The Chicago Neurological Society was founded in 1894, and other societies in St. Louis, Baltimore and elsewhere were organized at later periods. The American Neurological Association was founded in 1875.

At the twentieth anniversary of the founding of the Society, held in January of 1904, Dr. Mills read an address on Neurology in Philadelphia from 1874 to 1904. In this paper he gave a résumé of the work of the society during the first twenty years of its existence which I might be permitted to quote in this connection.

"During the twenty years of the existence of the Philadelphia Neurological Society 616 contributions to its proceedings have been made. The presentation of a list of these contributions would probably serve no useful purpose except to show the interest and industry which have characterized the society during its life of two decades. A tabular analysis of these contributions may be of some value as giving a general idea of the work done. A list of the contributions was prepared under my direction by Dr. J. W. McConnell and Dr. T. H. Weisenburg. I have found it somewhat difficult to group these contributions under a few heads, but with the list before me and with a personal knowledge of the work of the society, from whose meetings I have been absent less than a dozen times since its foundation, I have arranged these contributions to the proceedings under the four heads of (1) formal papers; (2) minor scientific contributions; (3) cases reported and patients presented; and (4) pathological specimens and special pathological reports. Under formal parts have been classed all contributions important in their quality or length or in both; under minor scientific contributions have been included notes on special matters often of much value, such as those referring to new instruments or methods of investigation, new drugs or therapeutic measures, and new observations in symptomatology; under cases reported and patients presented have been grouped not only patients actually presented to the society, but also cases and sets of cases reported from time to time without presentation of the patients; and under pathological specimens and special pathological reports have, of course, been

classed the numerous specimens with and without special reports so frequently exhibited at the meetings of the Society. Thus classified, the 616 contributions to the Society's proceedings during the twenty years have been as follows: Formal papers, 244; minor scientific contributions, 39; cases reported and patients presented, 229; and pathological specimens and special pathological reports, 104; total 616.

"A study of this wealth of material shows that scarcely anything has been brought before the society which has not had some interest, and that some articles of great importance, and a few that may be classed as among the most valuable contributions to neurology during the last twenty years are to be found. During the twenty years about 140 meetings of the society have been held, so that the average number of contributions at each meeting would be between four and five.

"It is difficult to single out particular contributions from a society's proceedings of two decades for special mention, but a few of the most important of these are S. Weir Mitchell's edema in hysterical hemiplegia (1884); Dercum and Parker's paper on the artificial production of a hysterical state with convulsive manifestations (1884); the nuclear anatomy of the nerves which supply the muscles of the eyeball, by E. C. Spitzka (1888); Lloyd's traumatic affections of the cervical region of the spinal cord simulating syringomyelia (1894); a case of merycismus, by Riesman (1895); experimental lesions of the cortical tissues of the rabbit's brain induced by alcoholic poisoning, by Berkley (1896); gliosis cerebri, by Sailer (1897); J. K. Mitchell's headache with visual hallucinations (1897); the report by E. Lindon Mellus of original investigations on motor tracts in the brain and cord (1898); and Spiller's Pepper Laboratory contribution on a case of amyotrophic lateral sclerosis showing the extent of the motor cortex in man (1899)."

Following the plan of enumeration used by Dr. Mills in recording the work of the first twenty years of the society, an examination of the minutes of the scientific proceedings shows that during the last decade 568 contributions have been made. This record indicates that while during the first twenty years the average number of contributions for the year was about thirty or in the seven monthly meetings between four or five for each, in the last ten years the average almost doubled, being annually 56

or about 8 for each meeting. The analysis of these 568 contributions shows that 153 were formal papers, 14 were minor scientific contributions, 370 were cases reported and patients presented, and 31 were pathological specimens and special pathological reports. While the number has increased their value has not diminished.

During the last decade several important symposia and special meetings have been held. The first of these was a combined meeting of the society with the New York Neurological Society on November 24, 1906. At this meeting cases which had been chosen because of their unusual interest were shown by Drs. Burr, McConnell, Mitchell, Rhein, Gordon and Weisenburg, and an important paper was read by Dr. Spiller on "Psychasthenic Attacks Simulating Epilepsy." Dr. H. S. Fraenkel of Berne, Switzerland, was present and discussed several of the papers, especially that on tabes, giving particular attention to an exposition of his methods of treating ataxia by systematic exercise. The presentation of these cases preceded the main topic of the symposium which was a consideration of Marie's views of aphasia, which was discussed by Drs. Mills, Dercum, and Lloyd and by Drs. E. D. Fisher and M. Allen Starr of New York and Fraenkel of Berne.

On March 26, 1909, the distinguished bacteriologist of the Rockefeller Institute, Dr. Hideyo Noguchi, enlightened the society with a valuable demonstration of a new and simple system of the complement fixation test, and of the butyric acid test for syphilis and parasyphilitic affections.

A second combined meeting of the New York and Philadelphia Neurological Societies was held in Philadelphia, December 18, 1909, at which, in the first place, a series of cases was shown by Drs. Potts, Mitchell, McConnell, Dercum and Allen. In the second place, Drs. Spiller and Frazier contributed a paper with illustrative cases on the treatment of spasticity by resection of the posterior spinal roots and another article was contributed by Dr. Frazier on division of the auditory nerve for persistent vertigo and tinnitus. Dr. Mills introduced the special subject for discussion which was the clinical study of changes in sensibility due to organic and functional disease, especially with reference to the methods of testing. This paper was discussed by visiting members from New York and some of the members of the society.

On February 24, 1911, Dr. Sidney I. Schwab of St. Louis opened a discussion on the Freudian theories of the neuroses by an instructive paper entitled "An Estimate of Freud's Theory of the Neuroses and Its Value to the Neurologist."

A special meeting of the Neurological Society was held on Friday, November 10, 1911, in honor of the Congress of Clinical Surgeons of North America. At this meeting the following program was presented:

Dr. Hugh T. Patrick of Chicago: "The Technique and Results of Deep Injections of Alcohol for Tic Douloureux." Dr. Harvey Cushing of Baltimore: "Some Clinical Types of Disordered Function of the Pituitary Body," Dr. A. R. Allen of Philadelphia: "Operative Treatment of Experimental Lesion of the Spinal Cord Equivalent to the Crush Injury of Fracture Dislocation of the Spinal Column."

On February 27, 1914, at a special meeting for the discussion of aphasia papers were presented by Drs. Mills, Dercum, Burr, Lloyd, Rhein, Spiller and Weisenburg. The older or classical views of aphasia, Liepmann's doctrine of apraxia, the relations of aphasia to mental disorders and the special significance of anarthria were leading subjects of discussion, and Dr. Spiller presented specimens from the Laboratory of Neuropathology of the University of Pennsylvania showing lesions causing aphasia situated at various positions in Wernicke's zone, the lenticular zone and Broca's convolution.

The papers and discussions of all the symposia referred to were of a high order. The society held special return meetings in New York and Boston in combination with the New York Neurological Society and the Boston Society of Psychology and Neurology.

Time will not permit me to review in detail the contributions of individual members of the society to some of which however I would like to make brief reference. There is no doubt that in some instances the omissions will be quite as important as the inclusions; indeed I have found it difficult to discriminate as to what should be recalled, owing to the wealth of material at my disposal.

Dr. William Pickett, whose loss is sadly remembered by the society, shortly before his death presented several careful psychiatric and neurological contributions, including an elaborate

statistical and general study of puerperal insanity based on work at the Philadelphia Hospital for the Insane.

Dr. John K. Mitchell among other valuable contributions gave to the society studies of some phases of tabes and recorded a number of cases of much diagnostic and therapeutic importance.

Dr. C. W. Burr in this period of ten years has presented besides several formal papers a series of instructive and in some instances unique cases, among which were cases of epilepsy with myoclonus, paralysis agitans in the negro, tetany involving one side of the body, and mirror writing.

The variety and value of the contributions to the proceedings of our society by Dr. William G. Spiller are such that it is difficult to properly recognize these in a brief retrospect. Twenty-five papers and twenty cases were presented by him during the last decade, he contributing also numerous pathological specimens.

A few among his formal papers of special interest were those on multiple motor neuritis (with Dr. Longcope), the clinical resemblance of cerebro-spinal syphilis to disseminated sclerosis (with Dr. C. D. Camp), the syphilitic form of multiple sclerosis, dissociation of sensation in pontine lesions, the treatment of spasticity by resection of the posterior spinal roots (with Dr. Frazier), and traumatic brachial neuritis, probably caused by tearing off the nerve roots; while among the interesting cases which he presented were one of polioencephalitis superior and inferior involving the corpora quadrigemina, one with bilateral spasticity and athetoid movements, one of hemicraniosis, and one of dystonia musculorum deformans.

Notable among the contributions of Dr. C. S. Potts have been those on pseudo-sclerosis (with Dr. Spiller), and cases of traumatic cervical hematomyelia, periodic paralysis, tic of the tongue, and encephalitis due to gasoline poisoning.

Dr. D. J. McCarthy, from his rich experience at the Phipps Institute and elsewhere, contributed to our proceedings papers and cases dealing with the various phases of tuberculous involvement of the nervous system and other contributions by him worthy of note were on ascending paralysis, psychoanalysis considered apart from the Freud concept, and the hemosiderin infiltration of the wandering cells of the pia-arachnoid, the last presenting a subject of rare interest.

Instructive and interesting cases were presented by Dr. J. W.

McConnell including a case of postero-lateral sclerosis with involvement of the cells of the anterior horns, one of voluntary monolateral ocular movement, and one of aphasia and apraxia with unusual associated movements. A specimen exhibited by him showing the limitation of the face area in the human cortex was a distinctly valuable contribution to accurate cerebral localization.

The list of contributions by Dr. Alfred Reginald Allen includes annular degenerations of the spinal cord, the distribution of the sixth cervical anterior root in the cervical spinal cord, and a symptom-complex of transverse lesion of the spinal cord and its relation to structural changes therein. His presidential address on psychoanalysis in June, 1912, will also be especially recalled.

Dr. John H. Rhein's paper discussing apraxia in relation to aphasia will be remembered by members of the society as one of the most valuable contributions to the subject to which Liepmann has given so much attention. This article since its publication has been much cited both here and abroad. A paper by Dr. Rhein on spastic paraplegia is also worthy of special note, and his list of cases presented illustrated a variety of interesting or unusual clinical types.

The presidential address of Dr. George E. Price, June 23, 1914, on a "Sixteenth Century Paranoiac" was as entertaining as it was instructive and represented work in a field not much tilled by members of the society. To Dr. Price the society also owes the discussion of two forms of familial disease—one of migraine in a large family associated with unusual sensory disturbances, and the other, spinal gliosis occurring in three members of the same family.

Papers by Dr. S. D. Ludlum on neurofibrillary changes, on regeneration of the peripheral nerves and various serological contributions in collaboration with Dr. E. P. Corson-White, have greatly enhanced the value of our proceedings.

Worthy of particular mention are the papers and cases of Dr. Williams B. Cadwalader on a case of segmental astereognosis resulting from a lesion of the parietal area, the pathology of acute anterior poliomyelitis, a case of unilateral optic atrophy and contralateral hemiplegia consequent upon an apoplectic attack, and pituitary and uncinate symptoms and lesions (with Dr. Mills).

The contributions of Dr. S. D. Ingham include encephalitis with a report of two cases with necropsy, and a case of cerebro-spinal syphilis with internal hydrocephalus causing symptoms of cerebellar tumor.

The record of cases of unusual interest in some instances is so large and has been added to by so many members of the society that it is difficult to make any discriminating reference to them. Dr. Alfred Gordon, for example, among other cases and contributions has reported a specially instructive case of neurofibromatosis, several cases of myatonia congenita, a series illustrating unilateral ascending and descending paralysis, bulbar palsy with total aphemia, a neuritic form of syringomyelia, and a case of hemiplegia with hemiatonia probably due to lenticular lesion. To Dr. Samuel Leopold the society owes the presentation of specimens of osseous plaques of the spinal pia-arachnoid with a discussion of their relations to pain in acromegaly, and cases of unilateral cauda-equina lesion.

Dr. N. S. Yawger's contributions on indurative headache and colloid bodies in the central nervous system will be especially remembered, the first involving consideration of a therapeutic measure of great value and the second as of much pathological interest.

The physiology of the cerebro-spinal fluid as a problem in intracranial surgery by Dr. Chas. H. Frazier was a comprehensive and excellent study of the nature and functions of the cerebro-spinal fluid.

Among the contributions of Dr. C. D. Camp, now clinical professor of neurology in the University of Michigan, were pre-eminently, a study of six cases of paralysis agitans and another paper on multiple sarcomata of the brain and cord (in collaboration with Dr. Burr). He also brought before the society a variety of cases, all of particular interest.

To two of our out-of-town members, Dr. Tom A. Williams, of Washington, and Dr. C. M. Byrnes, of Baltimore, the society is indebted. Among the interesting cases presented by the former should be mentioned variable migrainous recurrent paralyzes followed by permanent lateral homonymous hemianopsia, cerebellar disease and bulbar thrombosis resulting from malaria, and interesting cases in which the value of psychoanalysis was dem-

onstrated. Two important contributions of Dr. Byrnes embodied an interesting description of a new instrument and method of directly injecting the gasserian ganglion for the relief of pain in tic douloureux and an account of a new method of injecting mercury directly into the spinal canal for the treatment of syphilis.

THIRTIETH ANNIVERSARY DINNER

The dinner in celebration of the thirtieth anniversary of the society was held at the Philadelphia Art Club, November 28, 1914, at 7:30 o'clock. Sixty-six members and invited guests were present. In accordance with the program arranged responses to toasts were first made by Dr. George W. Jacoby, of New York, president of the American Neurological Association, by Dr. Morton Prince, of Boston, by Dr. Hugh T. Patrick of Chicago, and by Dr. Henry M. Thomas, of Baltimore. In addition to these responses remarks were made by Dr. George E. deSchweinitz for the College of Physicians of Philadelphia in the absence of Dr. James C. Wilson, president of this association, by Dr. William Duffield Robinson, president of the Philadelphia County Medical Society, and by Dr. S. D. Risley, president of the Philadelphia Medical Club. Dr. Graeme M. Hammond of New York also spoke in response to a call by the president. A letter was received from Dr. William L. Rodman, president-elect of the American Medical Association, expressing his regret that an unexpected call out of the city had prevented him from being present at the dinner as he had expected. In his letter he extended his congratulations to the society, expressing his appreciation of its high position in American medicine.

THE AMERICAN NEUROLOGICAL ASSOCIATION

BY GEORGE W. JACOBY, M.D.

The present world war has cut so relentlessly and so deeply into all conditions of life, even into those that are apparently unrelated to it, that it may well serve as an introduction to remarks upon any subject whatsoever. Every conversation at this time of unexampled stress and affliction turns about the war, starts from or ends with it. For us, as medical men, there are in this state of affairs yet other considerations, aroused by the reflection that it is the task of war to destroy human life while the prime object of all medicine is to preserve it, and that science in general must be most seriously affected in so far as the international exchange of ideas, that most powerful impetus to scientific research, has been brought to a complete standstill. This of course can be but temporary, for no matter how much political and economic interests may collide, science is and must remain international. But while we are waiting for a reestablishment of international relations, scientific investigation must keep on. Scientific work can find no respite for it represents the true neutral domain upon which all partisan strife must cease and upon which alone the logic of facts can count. True science is not the one that invents machines of war and furnishes nations with instruments of destruction, but the one that unites the noblest and the best minds in the common endeavor to exterminate ignorance, prejudice and superstition, the worst enemies of progress, and to do so not with mines, airships and 44-centimeter guns, but with the weapons of the intellect.

It is to such warfare we have devoted our lives and it is as representatives of such warfare that I address you to-night in behalf of the American Neurological Association.

Gentlemen, that association founded in 1875 was also the scene of many a stormy battle. In fact, its very baptism was a troublous one, for no sooner had it elected S. Weir Mitchell, of Philadelphia, as its first president, than for some occult and never to be disclosed reason, he declined to accept the office and J. S.

Jewell, of Chicago, was selected the standard-bearer of the association. Having secured a president, the association determined to retain him and Jewell was reëlected and reëlected until he had served for four full years. During those first years, I have been told, and subsequently, I know, the association proceedings were of the liveliest and frequently of the most belligerent nature. The membership then was small, for of the 35 who accepted the invitation to be present on the 2d, 3d and 4th of June, 1875, the first meeting of the association, but twenty came and only sixteen names are to be found as original members of the association. Of these, three came from Boston, three from New York, two from Baltimore, two from Philadelphia and one from Albany, Brooklyn, Chicago, Kankakee, New Orleans and Washington.

I shall refrain from developing the history of the association, for such a history would mean giving a history of the progress and growth of neurology itself. Most of the genuine original work in neurology done in the United States during the past forty years has been first presented to the American Neurological Association for criticism and endorsement, and the names of almost every American worker in neurology who has contributed anything of worth, will be found represented on its list of membership. In other words it is in the truest sense a national association of specialists. Nor shall I individually mention the names of those who have been most prominent in the association's work, for should I do so, I fear you would think I was undisguisedly and broadly trying to heap encomiums upon the city of Philadelphia.

Many changes have naturally taken place in the association since it was my good fortune to be admitted as a member. Energetic young workers have come in by the score and of the older ones many have paid their debt to nature and left us to give battle alone.

A long list of dead today forms the honor list of our association. Men whom we should never forget and should always recall with gratitude, for it is their shoulders that serve us as a point of vantage from which to enlarge our horizon. Of these I do want specially to mention the names of three men in whose work all speculation, whether philosophically or theologically colored, was *a priori* excluded and whose device was "exact investigation and facts." How different from some neurologic

fantasts of recent years. These are J. S. Jewell, Thomas A. McBride and William R. Birdsall.

Jewell, of Chicago, the first president of the association, was but fifty years old when he died in 1887. He was an indefatigable worker, a pioneer in many ways, one of the founders of the *JOURNAL OF NERVOUS AND MENTAL DISEASE* and for years its chief editor. No one who was present at the meeting of the association he last attended can forget his brilliant mind, his charm of manner and conversation and the contrast furnished by his emaciated body.

McBride died in 1886 at the age of forty-two. He was a man of the kindest and most generous disposition, of a broad liberal scientific spirit, and one in whom was most happily blended the qualifications of a general clinician and neurologist.

Birdsall was above all my intimate personal friend. Our lives were closely intertwined in Vienna, Paris and New York. His unselfish devotion to everything that seemed noble and true have enshrined him in the memory of all those who knew him well. His brain was that of a scientist, his heart that of a child.

But enough of persons and facts. There is and always has been another phase of character, that seems to me to be individual to the American Neurological Association and that is its capacity for social enjoyment. What Pericles said to the Athenians I might say of the American Neurological Association. He says: "Our laws have further provided for the mind most frequent intermissions of care, by the appointment of public recreations, elegantly performed with a peculiar pomp, the delight of which is a charm that puts melancholy to flight."

If there be any here to-night who have not taken part in one of the association's social functions, I can but say, make good your loss, for, as Kipling says:

When you grow older and skin your shoulder
At the world's great wheel, in your chosen line,
You'll find your chances as time advances
For taking a lark, are as slim as mine.

Now Mr. President and gentlemen, let me bring these remarks to an end by expressing to you my great pleasure at having been honored by this invitation to be with you to-night and as spokesman for the American Neurological Association, to call to the Philadelphia Neurological Society an "*Ergo Bibamus*."

AMERICAN NEUROLOGY OF THE PAST—NEUROLOGY OF THE FUTURE.

BY MORTON PRINCE, M.D.

In speaking of "American neurology of the past" one's thoughts naturally turn back to the successive problems of the times and to the men who were occupied with their solution. I have been exceedingly interested in looking over the early Transactions of the American Neurological Association and noting the differences in the problems which came up from time to time and occupied the thought of each epoch. Each epoch has its own problems, but those problems are brought to attention by the discovery of new facts giving a new viewpoint of old problems hastily assumed to be settled; or by the realization of truths forced upon us by changing conditions, or by needs to which previously we were oblivious. Each new discovery in science opens, as it were, the gate to a new field into which rush the explorers who proceed to search in systematic manner every inch of the soil for golden nuggets or precious stones of knowledge. And then, that field exhausted, there is a pause until a gate to a new field is opened by some pioneer, and again there is a rush of explorers to discover what previously unsuspected truths may there be found. Necessarily there became ranged about the problems of an epoch the names of those master minds, of those inspired by the call of discovery, who contributed to their solution. I say "contributed," for rarely if ever has any one man completely solved any problem. Indeed no problem is ever completely solved in the true sense. It is with these considerations in mind that I would review, however, cursorily, "American neurology of the past."

The American Neurological Association was founded in 1875—forty years ago coming next spring; *i. e.*, ten years before the founding of your society. As you run over the names of the original thirty-five members I think you must be struck with the fact that, with a few notable exceptions like W. A. Hammond,

J. J. Putnam, Webber, Seguin and Hamilton, the membership was not one of specialists, men who devoted themselves exclusively to neurology: it was in the main rather of internists who had a greater or less interest in neurology. Indeed some of the members had acquired their chief distinction in the practice of general medicine or in other fields. Among these I may mention the well-known names of Pepper and H. C. Wood, of Philadelphia, Edes and Edward H. Clarke, of Boston, Hun, of Albany, Dalton, of New York, and Miles of Baltimore. Correspondingly the papers, with important exceptions, took on rather a clinical and theoretical type during the first few years. Nor is one impressed with the problems as being of very profound significance.

Gradually, you will notice, there was an influx of young men who devoted their activities wholly or most intensively to neurology until, about the date of the founding of your Philadelphia Society thirty years ago, there had arisen a group of thoroughly trained American neurologists. It seems to me therefore fair to say that American neurology, or at any rate the beginning of the great advance in neurology in America was coincident with the founding of your society.

The theme then, of which I would sing is of the problems of neurology in the twenty-odd years long epoch, beginning with the early eighties, and of that group of pioneers who contributed to their solution. This is all the more a pleasing theme because it takes us within the sphere of activity of the oldest and older of those still in harness, many of whom I see here present tonight.

If we would view this group of early American neurologists and their problems, in true perspective, we must remember that at a preceding epoch, not so very long antedating this one, neurology was necessarily largely clinical and theoretical from lack of anatomical data. It was a *clinical and theoretical school*. Close observers of the clinical manifestations of disease, the neurologists of this school, were obliged in the existing undeveloped state of normal and pathological anatomy, to rely to a large extent upon speculative reasoning to furnish the underlying pathology. And yet, trained to think profoundly, they often forecast the future objective demonstrations of later schools by pure reasoning. Thus Hughlings Jackson may be said to have laid down the *principle* of cerebral localization on theoretical grounds.

Then the rise of the great anatomists, represented by Stilling, whose work will always remain a classic, and Meynert, not to mention other notable contributors, made possible the succeeding school which sought systematically to correlate clinical observations with normal and pathological anatomy. This school may be termed *clinico-anatomical*. It was, of course, a tremendous advance. The discoveries in the anatomy of the nervous system had given a new aspect to old problems, and had opened new problems which before were not thought of. At the same time the investigations of this school contributed enormously to our knowledge of normal and pathological anatomy and of the physiology of the nervous system. Its method was the only method by which the problems of organic neurology could be scientifically approached.

Of the more illustrious of this school I may mention only a few. The names of Charcot, Duchenne, Westphal, Leyden, Kusmaul, Erb, Ferrier, Hitzig, Buzzard, Bramwell, Strümpell, and Friedreich, Weir Mitchell and Hammond, stand out in bold relief, though others might with equal justice be mentioned.

Although neurology belongs to internal medicine, the almost unbroken character of the ground at that early period, and the complexity of the problems presented, required intensive study and therefore men who at least specialized in this field. It is interesting to note that many of the men I have mentioned who developed neurological "kultur"—and it is true of others—were general internists but men who gave their most intensive energies to neurology. They were mostly clinicians, but the problems they solved were more than clinical; they were pathological, physiological, anatomical, etiological, and surgical, and, to an extent, psychological. This is true in a measure, I think, of the whole of internal medicine but it was more peculiarly true, I think, of neurology of that period. Thus there was developed to meet the needs, a group mostly of Europeans of great brilliancy; but that group had worthy successors in the brilliant group of American neurologists of the same clinico-anatomical school but of the next epoch to which I will presently come.

At this time Weir Mitchell and the elder Hammond, who have given us worthy successors in their sons, were both at the height of their fame. Mitchell had done his classic work on "Injuries

of Nerves" and his treatment of neurasthenia had spread through Europe as well as America and everywhere was in vogue. Hammond's text-book was the accepted text-book on nervous diseases in our colleges, and remained about the only thing in English until the youthful Hamilton produced his work. Then there was Beard who had recognized the functional character of a medley of theoretical organic diseases, rivalling in their multiplicity and variegations the races making up a European empire, and unified them in one general functional concept *neurasthenia*. Spinal congestion, spinal irritation, cerebral congestion and anemia, "congestion at the base of the brain," then lost their autonomy and were wiped from our neurological slates. He also recognized the phobias as at least symptom complexes.

Then there were amongst the earlier men who are not with us now, Spitzka, keen as a rapier and unrivalled as an anatomist; and Seguin by whose early death neurology lost one of the ablest minds and most accurate and thorough observers. He with Shaw and Miles, in 1877, was the first to contribute to cerebral localization.

I did not become a member of the Association until late in the eighties: then I found that there had preceded me that group of neurologists, my contemporaries, most of whom are still with us.

As I look back now to that early American epoch there is one thing that strikes me as rather remarkable and that is the youthfulness of the majority of that group. They seem to me now as having been at that time mere youths, and yet nearly one and all had already attained an enviable reputation—some had reached eminence. I doubt very much if that group, in this respect, can be paralleled in any other field of medicine.

It will be interesting now to take a glance—but it can only be a glance—at some of the problems which sprang up in the night, as it were, during this later clinico-anatomical period and with which the names of American neurologists have become honorably associated. As these problems of the past come to my mind many names arise too, but my memory is guided simply by the reminiscent mood of the moment unaided by any attempt at search of the literature. Undoubtedly my memory will miss many names which should be recalled.

First and foremost there were the great problems of cerebral

and spinal localization. These sprang at once to the front, stimulated by the epoch-making researches of Ferrier and Hitzig carried on by the experimental method on the brains of animals. It became the task of neurology to confirm the evidence in man and extend our knowledge by clinical and pathological data.

And then there followed the problems of surgical operations on the brain and spinal cord, to which Horsley gave the first impetus by his original and daring operations on the brain. To the solution of one or more of these problems many American neurologists have contributed, but I am sure that all will rejoice to recall the names of Mills, Starr and Dana as pioneers and the most systematically engaged at this epoch with these problems, Dana more especially in connection with sensory localization. We are all glad to acknowledge in particular our indebtedness to your president, Dr Mills, who, still animated by the persistent and unabated enthusiasm of his youth, has never ceased to make enlightening contributions. I said "systematically engaged," for the important contributions of Seguin, J. J. Putnam, Knapp, Lloyd, Walton, Dercum, Collins, and Spiller should not be forgotten; nor, I doubt not, those of others. Your Spiller seems, like Socrates, to have constituted himself a neurological busybody and concerned himself with about everybody else's problems as well as his own and illustrated them all.

The recognition and differentiation from myelitis of *multiple neuritis* was another problem of this epoch. Then there were the *dystrophies*, as distinguished from the spinal atrophies; the *syphilitic etiology of tabes* originally enunciated, I think, by Erb; *myxedema* as a glandular disease; *syringo-myelia* and the varieties and pathology of *aphasia*. To each of these there were many American contributors and I should fear to particularize.

We older fellows remember the hectic war waged over the pathology of the traumatic neuroses. This apparently inoffensive and innocent little scientific problem had been as a very Balkan question to the peace of neurology, arraying brother against brother, even unto a titanic struggle between those two renowned pacifists whom I need not mention, not only in what—by a euphemism—might be called scientific discussions, but in the hardly less polemical expert arena of the law courts.

It was largely the influence of the French and American schools that gave us our modern functional conception of these

neuroses. With this conception J. J. Putnam and Walton are closely associated as pioneer contributors and exponents, while Knapp was persistently engaged with the enlargement of our knowledge of the symptomatology and of the hotly contested prognosis (when he wasn't engaged in battling with Walton.)

With *adiposis dolorosa* one name alone comes to mind—that of Dercum, who enjoys the enviable distinction of priority of discovery. Obviously I cannot review all the problems of the period and the work of those engaged thereon, but I cannot pass over those of Little's disease and amaurotic family idiocy, for the elucidation of which we are deeply indebted to Sachs.

With the discovery of *diffused sclerosis* in *anemia* and *debility* the names of J. J. Putnam and Dana will always be associated. I pass over family periodic paralysis, which Taylor has so well illuminated, the various problems raised by Graves' disease, and even the great contributions of Cushing to diseases of the hypophysis cerebri. These last belong to a succeeding epoch. The younger men who will succeed their sires must wait for their own historian, I'm sure none shall say, apologist, who will reminisce as I have done.

NEUROLOGY OF THE FUTURE

What of the future? Have all the great problems of neurology been worked out? So some of our pessimistic confrères, who sometimes give us the blues, would have us believe; and are we prepared to hand over the field as fully tilled to general internists? This is surely a superficial point of view. There always will be problems requiring the intensive study of specially equipped investigators. No problem is ever or can be ever completely worked out. New data require new points of view, old accepted explanations have to be revised in the light of new knowledge, newly perceived relations examined. Often when a problem has been accepted for a long time as solved, we lose sight of the fact that what we accepted as a solution was only an interpretation, a theory, and not a fact; and in the light of new data the theory must be discarded and a new explanation found. Witness the once accepted theory of the transmission of phthisis by direct heredity as affected by the discovery of the tubercle bacillus and that of "laudable pus," which many of us here were taught before Lister's discoveries.

In neurology, take the theory of the parasymphilitic pathology of general paralysis and tabes as affected by the discovery of the spirochete in the tissues—what a problem this opens in the field of therapeutics! Marie's bomb tore to tatters our previously accepted schematic conception of aphasia but has left us with only crude, inadequate theories of function. Here is a problem for the future of a most intense interest. Our knowledge, perhaps, satisfies, though not wholly adequately, the practical needs of clinical diagnosis but not of brain physiology. It is really a psychological problem. I have yet to read of a case adequately studied in view of the requirements of the problem.

I suppose I shall shock every dyed-in-the-wool "localizationist" if I suggest that even the present doctrines of the cerebral localization of functions must be regarded as crude and provisional in many respects. I mean so far, at least, as they pretend to locate psychological and even physiological "functions." If I am right the problems will have to be completely restudied in view of the inconsistencies of accumulated data and a newer conception of brain physiology. As von Monakow has pointed out, cerebral localization is a clinical conception rather than a psychophysiological or functional one.

It is enough simply to point to the almost wholly new problems that have sprung to life by the discoveries in bacteriology, serum pathology and the parts played by internal secretions. When one thinks of the almost unknown laws of heredity and of the relations of mental defects to crime, what a multiplicity of practical problems awaiting future solution crowd into the mind!

CLINICO-PSYCHOLOGICAL SCHOOL

I have thus far spoken only of those past and future problems which can only be solved by clinico-anatomical methods. But there are pressing problems in another domain which, if I were guided by tactfulness, I would approach on this occasion with hesitation because their agitation seems to have the singular effect of disturbing the mental peace of this good City of Brotherly Love. Methinks I hear now the distant rumblings of the coming storm! I refer—you already guess it—to the problems of functional neurology and the psycho-neuroses which are to become some of the important problems of the future.

Unfortunately this field has been, until recently, strangely

neglected; I would be within the bounds of moderation if I said, shunned. I said "unfortunately" and "strangely" because fully 75 per cent. of all the nervous ills which torment and destroy the happiness and well-being of humanity, as represented in neurological clinics, belong to the functional category. I forbear to dilate upon what is obvious even to a layman, that the disregard of the study from the functional point of view, of these affections, by trained neurologists, has left the field to be explored therapeutically, and therefore, pecuniarily, by charlatans, mind-curists, Christian Scientists, osteopaths, and all the various therapeutic sects who have found the failures of the medical profession a rich harvest for their own profit.

The reasons of this deplorable short-sightedness I will not go into beyond mentioning the difficulties of the problems and the necessity of leaving the anatomical viewpoint.

A new clinical school of research has invited the activities of many neurologists, psychiatrists, and internists who have clearly recognized that clinico-anatomical methods are as absolutely inadequate to solve the riddles of the psycho-neuroses as they are of normal psychology. This new school, in contradistinction to the clinico-theoretical and the clinico-anatomical, may be termed the *clinico-psychological*. You younger men, with your elastic minds, your energies unabated by time, your scientific curiosity undulled, will take up these problems of the psychoneuroses by the new methods and from the vantage point of the new knowledge.

Obviously, when the problems by their very nature do not admit of one avenue, or mode, of approach, another must be found, even if it be less easy and congenial. I remember a good friend and classmate of mine, a member of the A. N. A., once said to me "Drop your investigations into hysteria. Hysteria has been the grave of every neurologist who has attempted to study it, including Charcot." He may have been right, but it is in order, I think, to remind him that Charcot's fame still lives, and I can show him the liveliest group of corpses that he ever set eyes on. Another distinguished neurologist and member of your Society, for whose attainments I have the highest respect, wrote me, I must believe, out of the kindness of his heart, that "studies of this kind can be better done by a policeman or a detective than by a gentleman."

Psycho-pathology seeks to unravel the complexity of motives or motivating forces, if you prefer, which determine mental behavior and to follow their impulses to their last effects in the human organism. It would be as useless to try to explain such problems by the use of the anatomical method as it would be to seek to explain, why Smith hates Jones or is jealous of Brown, by a congested optic thalamus. Some irreverent wag might be brutal enough to remind you that the real reason is because Jones punched a hole in Smith, and Brown had robbed him of his best girl.

Will you say I am untactful if I remind my anatomical friends that even if a hypothetical toxemia be accepted on theoretical grounds as a fundamental factor in dementia-præcox and manic-depressive insanity, that toxic principle in no way explains why a given demented person, for instance, thinks his body is made of glass or why the melancholic individual thinks he has committed the "unpardonable sin." Surely, the toxic explanation is as inadequate as it would be to explain the symptoms of tabes by the mere fact of infection by the spirochete.

For all such problems you will require as a fundamental prerequisite a knowledge of that great functioning mechanism, the subconscious. Their solution will require the discovery of the modes of activity by which it determines mental and bodily behavior. The subconscious may be likened to the works of a highly complex clock. These works are hidden out of sight. We are aware of the sounds of the chimes, the movements of the hands, of the procession of the signs of the zodiac, and the days of the month and of all the other accessories, but nevertheless it is the hidden works which determine these revealed movements that constitute the behavior of the clock.

Of late we have heard a great deal of German "Kultur." Well, there is a special World culture belonging to each of the sciences and which every scientist must attain in his own branch if he would be admitted into the "Society of Scholars." An important part of neurological culture of the future will be an understanding of the subconscious. Soon every neurologist will seek at least a working mastery of it. This culture already has permeated and strongly influenced the psychology of the universities, though, curiously enough, its chief source is to be found in the investigations of medical men.

The problems of the psychoneuroses are the problems of the subconscious. The subconscious is the great problem of the future that awaits you. This you younger men will take up and I doubt not you will solve it despite its perplexities and the difficulties of finding the true interpretation of its phenomena.

This is the message of the future I bring you—this is the gospel of culture I preach!

NEUROLOGY IN THE WEST

BY HUGH T. PATRICK, M.D.

Mr. Toastmaster, Members of the Philadelphia Neurological Society and Guests: You don't know it but your toastmaster has just given a beautiful demonstration of his psychotherapeutic skill. Ever since the scientific meeting last evening I have been suffering with an acute and violent attack of what they call in Boston symposiarchophobia and in Pittsburgh logophobia postprandialis, but his kind and tactful introduction has practically cured me. To be sure I am liable to relapse any minute but for the time being he has made me feel quite like another little country mouse of which you may have heard. The clinical history runs like this:

A farmer had spilled a little pool of whiskey on the barn floor. After he went away a mouse crept out, took a sip, scampered back to his hole and thought. Presently he came out, took another sip, went back and thought some more. Then he came out and took a big drink. Then he hopped upon a peck measure, stood up on his hind legs, bristled up his whiskers and said, "Now bring on that damned cat."

So, being pleasantly intoxicated by Dr. Mills' gracious words, I can proceed without intention tremor. First, I wish to express my sincere thanks for the kind invitation which allows me to be here this evening. I am proud and happy to bear from the west to the Philadelphia Neurological Society hearty greetings for the present, sincere congratulations for the past,—and for the time to come the confident hope that your younger and coming generations may not only prove worthy of their golden legacy but may make the future of this great society even more glorious than its past.

As to neurology in the west. Well, we sense the feelings of the old colored man who was sitting in the sun by the grocery door when a man stuck his head out and said, "Uncle, can you give me change for a ten dollar bill?" "No boss, no suh, I can't change no ten dollar bill but I do thank you fo' the

compliment." To think that you assume that there is a "neurology in the west" is most agreeable. It has a lovely sound. But it must be confessed that our neurology may be symbolized by something like the shadow on an adolescent's upper lip,—the substance of things hoped for, the evidence of things not yet seen. While neurology in the west may be spelled with the same letters as in the east it is writ exceeding small. Our infant feet are still trustfully toddling where you lead. We all know that westward the star of empire takes its way,—but sometimes like Tam O'Shanter it seems to be mighty reluctant to start; especially the star of neurological empire. That still hangs low in the east and we have no reason to suspect that in the immediate future the neurological guide posts will be pointing west.

The contrast between eastern and western neurologists may be expressed in different ways. We of the west are perhaps a little like some of the blue-stocking yankees who have come out to us. They still have the New England conscience but with western adjustments. We may be said to resemble eastern neurologists with western adjustments; sometimes a good deal more adjustment than neurology.

Your neurology of 25 years ago is remarkably like ours of today. Yours of today may be ours of tomorrow. At any rate, we like to think that the western neurologist is not a class, much less a genus, possibly not even a species, scarcely a variety; simply an ephemeral form due to transient environment and though not having your erudition, still having the three essential B's: that is, blood, brains and backbone of the eastern 1915 model.

There are still other contrasts. Neurologists, like society, may be divided into the observed, or responsible and observers, or irresponsible. We of the west belong in the latter category which is much less important but, I sometimes think, quite as much fun as the former. I wonder if you responsible observed ever say to yourselves

O, wad some power the giftie gie us,
To see ourselves as those chaps see us.

I suppose you never do but if you do and if some power would, I know you would be pleased. We are perfectly sure, as O. Henry says, that you are "advised about all the branches of information

contained in learning." You may have noticed that like the confirmed visitor of art galleries, we have developed a sort of mental torticollis from constantly looking up to you. And still at times you are beautifully human. Occasionally the luster of your culture is dimmed by pedantry and once in a while we seem to detect a little intellectual tenesmus. One or two of your longer articles have made me think of the experience one of my patients had with a colored waitress who gave notice. The mistress didn't want to lose her and said,—“Why, Eliza, haven't we always treated you well?” “Yas'm, but in dis yeah family dey am too much shiftin' ob de dishes fo' de fewness ob de vittles.” Some of these large literary vessels carry but little meat. And once,—I think only once—an abstruse lucubration of esoteric theorizing and recondite philosophizing reminded me of an incident at the old tanyard in my native village where we boys used to gather to tumble on the tan bark. The owner, for a sign, had bored a hole in the wall near the door and stuck a calf's tail into it. One day he noticed a man looking doubtfully at his sign but he didn't go in. So the tanner came out and inquired, “Want to buy some leather?” “No.” “Got some hides to sell?” “No.” “Are you a tanner?” “No.” “Are you a farmer?” “No.” “Well, what do you want?” “I am a philosopher and for half an hour I've been trying to figure out how the calf got through that auger hole.” Who wrote that abstruse paper? If you don't remember I shall not tell.

Neurologists may also be divided into producers and consumers. Here, again, we belong in the second class. But all consumers, like other fledglings, have appetites. And I venture to remark to you producers that just now some of us are hungering, perhaps quite unreasonably, for a better neurological technique. On surgical technique huge tomes have been written and are now in the writing. A good one on how neurologically to do things would be really satisfying pabulum. I do not mean that sublimation of the culinary art, laboratory technique; nor do I mean the technique of physical examination which somehow savors of the cleverness of a Sherlock Holmes on the trail of a lesion,—but a technique which will enable us to explore not a man's body but the man himself and to identify not his diseases but, what is of vastly greater importance, his disorders. Here is neurological territory almost unexplored. I think our cyclops-

eyed friends, the Freudians, are faithfully trying to get a squint at it through a gun barrel, but most of us, and most of you, pursue the beaten path and let the primeval wilderness alone.

In the neurology of out-west, then, there is much that is lacking, much that is primitive. But if we are embryonic in our attainments we are full-grown in our feelings and here in this city of brotherly love, with all my heart I tender to you of the east our admiration, our gratitude, our loyalty and, more than all, our personal affection.

NERVE DOCTORS AND OTHER DOCTORS

BY H. M. THOMAS, M.D.

It is with very real pleasure that I bring to you, Mr. President, and members of the Philadelphia Neurological Society, on this your thirtieth anniversary, the greetings and congratulations from your friends and admirers in Baltimore and the South.

I am but too conscious how inadequately my feeble words can express the debt of gratitude that we nerve doctors, working to the south of you, owe to you, Dr. Mills, and to your associates in this society.

If I may indulge for a moment in reminiscence, I was a medical student when this society was organized, not even a doctor, certainly not a nerve doctor. I became interested, however, and I well remember my enthusiasm a few years later in studying Dr. Mills's map of the cortical areas. He was a veteran of a hundred engagements even then, and since has never left the firing line. No change has been made in the boundaries of these areas without his help or his trenchant criticism.

As for myself, I have since been mostly occupied in reading the reports of the members of this society, and that my labor has been recognized is shown in my being asked tonight to respond to the toast "Nerve Doctors and Other Doctors."

My subject does not call for much elaboration. Nerve doctors are just like other doctors, except that they are better doctors, wiser doctors, and handsomer men. They would also be the busiest doctors if the other doctors could only see the light and refer to them all their patients who could best be treated by nerve doctors.

Although I have little fear of contradiction, yet there may be some one here so mentally and physically blinded, who, after contemplating this assembly and listening to the speeches, might say in paraphrase of Mark Twain's stranger's remark about Smiley's jumping frog, Dan'l Webster, "Well, I don't see no

pints about them doctors that's any better than any other doctors," and demand proofs of their vaunted excellences.

If such a one there be, I would remind him of our old friend, the German trombone player, whose assertion that he was the best trombone player in the whole United States, including Canada, was questioned. And I would say, as he said, with that confidence that can only come after a complete self-analysis, "Prove it? We don't have to prove it, we admit it."

There are nerve doctors and other doctors, and ordinary nerve doctors and nerve doctors Philadelphia style, and what a team you are! with Mills center, Dercum and Lloyd guards, Burr and Weisenburg tackles, Keen and Frazier at the ends, with Spiller giving the signals at quarter, and Jack Mitchell, McCarthy and Allen in the back field, and the side lines crowded with eager, seasoned alternates.

The members of this team know every style of play, but prefer to score by straight, hard, legitimate neurology, thus perpetuating the illustrious examples of their early trainers, H. C. Wood, and the lamented S. Weir Mitchell. They are not unmindful of innovations, and have started many and adopted some; and have even been known to use the somewhat risky, delayed forward pass of Freudian psychoanalysis, although they take particular delight when on the defence in breaking this play up.

The team is, indeed, in the All-American class, difficult to score against, and quite impossible to keep from scoring. May it never be weaker!

Philadelphia is justly celebrated for other things as well as for nerve doctors—for its boundless hospitality above all. On this auspicious occasion the Philadelphia Neurological Society has, as ever, welcomed its guests, has taught us good neurology, and now tonight has fed us on the best there is in the land. You, members of the society, have every excuse for satisfaction, and we, your friends, rejoice with you. We, too, are satisfied, indeed, so satisfied that I find myself like the frog, Dan'l Webster, after his forced feeding, quite unable to jump. Therefore after these well-intentioned, although ineffectual heaves and croaks, I, certain of your ready assent, shall stop, hoping soon to join you in the dreamless sleep of satisfied desire.

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MEMBERSHIP

Since the formal organization of the society in 1884 the total list of members up to November, 1914, has been 249. Of this number 35 have died and 91 have resigned.

ACTIVE MEMBERS (1914)

Elected

- 1900 Allen, Dr. Alfred Reginald, 2013 Spruce Street.
- 1912 Anderson, Dr. Ella Mary, Philadelphia Hospital for the Insane.
- 1907 Bloomfield, Dr. Maximilian D., 2008 N. Park Avenue.
- 1884 Bochroch, Dr. Max H., 1539 Pine Street.
- 1914 Bond, Dr. Earl D., 4401 Market Street.
- 1897 Boyer, Dr. H. P., 4602 Baltimore Avenue.
- 1883 Brubaker, Dr. Albert P., 3426 Powelton Avenue.
- 1903 Buckley, Dr. A. C., Friends' Asylum, Frankford.
- 1911 Burns, Dr. Michael A., 900 N. 48th Street.
- 1888 Burr, Dr. Charles W., 1918 Spruce Street.
- 1911 Byrnes, Dr. C. M., 207 E. Preston Street, Baltimore, Md.
- 1907 Cadwalader, Dr. Williams B., 1710 Locust Street.
- 1904 Carncross, Dr. Horace, 1003 Spruce Street.
- 1914 Clark, Dr. William L., 1809 Chestnut Street.
- 1911 Copp, Dr. Owen, 4401 Market Street.
- 1905 Cornell, Dr. Walter S., 1725 N. 16th Street.
- 1886 DaCosta, Dr. J. Chalmers, 2045 Walnut Street.
- 1886 Daland, Dr. Judson, 317 S. 18th Street.
- 1902 Darlington, Dr. Horace H., Concordville, Pa.
- 1914 DeLong, Dr. Percy, Cynwyd, Pa.
- 1884 Dercum, Dr. Francis X., 1719 Walnut Street.
- 1906 Donaldson, Dr. H. H., Wistar Institute.
- 1911 Drayton, Dr. William, Jr., 1316 Locust Street.
- 1914 Earley, Dr. Thomas B., Flanders Building.
- 1913 Easton, Dr. Flora Parker, State Hospital for the Insane, Norristown, Pa.
- 1904 Ely, Dr. Thomas C., 2018 Chestnut Street,
- 1910 Erney, Dr. Edwin H., 5602 Lansdowne Avenue.
- 1893 Eshner, Dr. Augustus A., 1019 Spruce Street.
- 1905 Frazier, Dr. Charles H., 1724 Spruce Street.
- 1906 Fussell, Dr. M. H., 2035 Walnut Street.
- 1904 Gilpin, Dr. S. F., 432 N. 52d Street.
- 1912 Golden, Dr. Louis M., 5035 Woodland Avenue.
- 1900 Gordon, Dr. Alfred, 1812 Spruce Street.
- 1912 Hallowell, Dr. Madeleine A., Vineland, N. J.
- 1897 Hamill, Dr. Samuel McC., 1822 Spruce Street.
- 1885 Hare, Dr. Hobart A., 1801 Spruce Street.
- 1904 Hawke, Dr. W. W., 218 S. 16th Street.
- 1905 Holloway, Dr. T. B., 1819 Chestnut Street.
- 1907 Ingham, Dr. S. D., 1831 Chestnut Street.
- 1912 Jackson, Dr. J. Allen, Phila. Hosp. for the Insane, 34th and Pine Sts.
- 1914 Jones, Dr. Isaac H., 1831 Chestnut Street.
- 1889 Keen, Dr. W. W., 1729 Chestnut Street.
- 1911 Krumbhaar, Dr. Edward Bell, W. Mermaid Lane, Chestnut Hill.
- 1909 Lane, Dr. P. H., 186 Bethlehem Pike, Chestnut Hill.
- 1911 Langdon, Dr. H. Maxwell, 2018 Chestnut Street.

- 1914 Leavitt, Dr. F. H., 1519 Pine Street.
 1907 Leopold, Dr. Samuel, 318 S. 15th Street.
 1884 Lewis, Dr. Morris J., 1316 Locust Street.
 1906 Lindauer, Dr. Eugene, 2018 Chestnut Street.
 1884 Lloyd, Dr. James Hendrie, 116 S. 21st Street.
 1903 Ludlum, Dr. S. D. W., 216 S. 15th Street.
 1899 McCarthy, Dr. Daniel J., 2025 Walnut Street.
 1891 McConnell, Dr. J. W., 701 N. 40th Street.
 1907 Meyers, Dr. Milton K., 1529 S. Broad Street.
 1884 Mills, Dr. Charles K., 1909 Chestnut Street.
 1912 Mitchell, Dr. H. W., State Hospital, Warren, Pa.
 1891 Mitchell, Dr. John K., 1730 Spruce Street.
 1886 Morrison, Dr. William H., 8021 Frankford Avenue.
 1892 Moulton, Dr. A. R., 5431 Locust Street.
 1907 Mulrenan, Dr. John P., 1228 S. Broad Street.
 1912 O'Brien, Dr. John P., 2733 E. Clearfield Street.
 1913 O'Hara, Dr. Thomas A., 227 S. 20th Street.
 1894 Ott, Dr. Isaac, Medico-Chirurgical College, Phila., and Easton, Pa.
 1900 Pepper, Dr. William, 1811 Spruce Street.
 1901 Peter, Dr. Luther C., 1527 Spruce Street.
 1913 Peterson, Dr. Jessie M., State Hospital for the Insane, Norris-town, Pa.
 1911 Phelps, Dr. George F., 6019 Lansdowne Avenue.
 1888 Potts, Dr. Charles S., 2018 Chestnut Street.
 1903 Price, Dr. George E., 1830 Rittenhouse Square.
 1911 Pritchard, Dr. W. C., 5614 Spruce Street.
 1895 Rhein, Dr. John H. W., 1732 Pine Street.
 1904 Rhein, Dr. Robert D., 2016 Pine Street.
 1894 Riesman, Dr. David, 1715 Spruce Street.
 1886 Robinson, Dr. William Duffield, 2012 Mt. Vernon Street.
 1892 Sailer, Dr. Joseph, 1830 Spruce Street.
 1897 Schively, Dr. Mary Alice, 318 Winona Avenue, Germantown.
 1886 de Schweinitz, Dr. George E., 1705 Walnut Street.
 1907 Sinkler, Dr. Francis W., 1606 Walnut Street.
 1911 Spencer, Dr. Elizabeth C., Bryn Mawr, Pa.
 1895 Spiller, Dr. William G., 21st and Chestnut Streets.
 1884 Taylor, Dr. J. Madison, 1504 Pine Street.
 1906 VanGasken, Dr. Frances C., 115 S. 22d Street.
 1902 Weisenburg, Dr. Theodore H., 2030 Chestnut Street.
 1911 Weiss, Dr. Benjamin P., 630 Spruce Street.
 1910 White, Dr. E. P. Corson, 216 S. 15th Street.
 1885 Wightman, Dr. J. G., 2030 Wallace Street.
 1886 Willits, Dr. I. P., 31 W. Walnut Lane, Germantown.
 1913 Wilson, Dr. George, 5000 Walnut Street.
 1886 Wilson, Dr. James C., 1509 Walnut Street.
 1901 Yawger, Dr. N. S., 2005 Chestnut Street.

NON-RESIDENT MEMBERS (1914)

Elected

- 1901 Allen, Dr. C. L., Los Angeles, Cal.
 1897 Barker, Dr. Lewellys F., Johns Hopkins Hospital, Baltimore, Md.
 1897 Berkley, Dr. H. J., 1305 Park Ave., Baltimore, Md.
 1898 Bennett, Dr. William H., Philadelphia and Atlantic City.
 1885 Brush, Dr. Edward N., Towson, Maryland.
 1884 Chapin, Dr. John B., Canandaigua, N. Y.
 1908 Davis, Dr. W. Price, Atlantic City.
 1902 Diller, Dr. Theodore, Pittsburgh, Pa.
 1884 Hinsdale, Dr. Guy, Hot Springs, Va.
 1911 McCafferty, Dr. G. W., Waymart, Pa.

- 1898 Mellus, Dr. E. Lindon, Brookline, Mass.
 1905 Orbison, Dr. T. J., Los Angeles, Cal.
 1897 Thayer, Dr. Wm. Sydney, 406 Cathedral Street, Baltimore, Md.
 1897 Thomas, Dr. Henry M., 1228 Madison Ave., Baltimore, Md.
 1910 Williams, Dr. E. M., Sioux City, Iowa.
 1908 Williams, Dr. Tom A., Washington, D. C.
 1896 Witmer, Dr. A. Ferree, Freeport, N. Y.
 1898 Winter, Dr. S. Elizabeth, W. Conshohocken, Pa.
 1908 Woods, Dr. Andrew H., Canton, China.

HONORARY MEMBERS

- *Charcot, Dr. J. M.
 Dejerine, Dr. J., 179 Boulevard St. Germain, Paris.
 Edinger, Prof. Ludwig, Frankfurt a/M.
 Erb, Prof. William, Univ. of Heidelberg, Heidelberg.
 Ferrier, Sir David, 34 Cavendish Square, W. London.
 *Gowers, Sir William R.
 Horsley, Sir Victor, 25 Cavendish Square, W. London.
 *Jackson, Dr. J. Hughlings.
 Marie, Prof. Pierre, 76 Rue de Lille, Paris.
 *Michel, Dr. Middleton.
 von Monakow, Prof. C., 116 Dufour Strasse, Zurich.
 Obersteiner, Dr. Heinrich, Billrothstr. 69, Wien.
 Tyson, Dr. James, 1506 Spruce Street, Philadelphia.
 Winkler, Dr. C., Heerengracht 501, Amsterdam.
 Wood, Dr. H. C., Chester Avenue and 42d Street, Philadelphia.

FOREIGN CORRESPONDING MEMBERS

- Babinski, Dr. J., 170 bis Blvd. Haussmann, Paris.
 Batten, Dr. Frederick E., 33 Harley Street, W., London.
 von Bechterew, Dr. W., Archijerejskaja Strasse, 6-8, St. Petersburg.
 Bianchi, Dr. Leonardo, University, Naples.
 Bramwell, Dr. Byrom, 23 Drumsheugh Gardens, Edinburgh.
 *v. Frankl-Hochwart, Dr. L.
 *van Gehuchten, Dr. A.
 Head, Dr. Henry, 4 Montagu Square, Hyde Park, W., London.
 Holmes, Dr. Gordon M., 58 Harley Street, W., London.
 Jendrassik, Dr. Ernst, Szentzirálya-u. 40, Budapest.
 Kappers, Dr. Cornelius Ubbo Ariens, Mauritskade 61, Amsterdam.
 Marinesco, Prof. G., Univ. of Bucharest, Bucharest.
 Mingazzini, Dr. G., Universitäts-Inst., Rome.
 Minor, Dr. L., Gusiaticnikoff 3, Moscow, Russia.
 Oppenheim, Prof. Hermann, W. Königin-Augustastrasse 28, Berlin.
 Petré, Prof. Carl, Lund, Sweden.
 Rossolimo, Dr. Gregoire, Skatetnoi pereouluk 24, Moscow.
 Rothmann, Prof. Max, W. Motzstrasse 89, Berlin.
 Schaffer, Dr. Karl, Budapest, Austro-Hungary.
 Stewart, Dr. Purves, 94 Harley Street, W., London.
 van Valkenburg, Dr. C. T., Univ. van Amsterdam, Amsterdam.
 Wilson, Dr. S. A. Kinnier, 14 Harley Street, W., London.

* Deceased.

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